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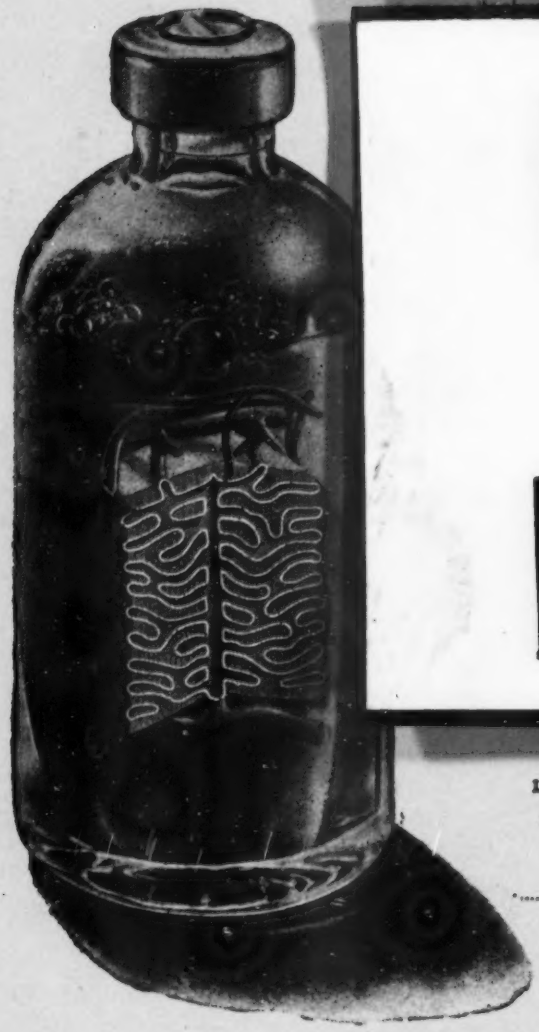

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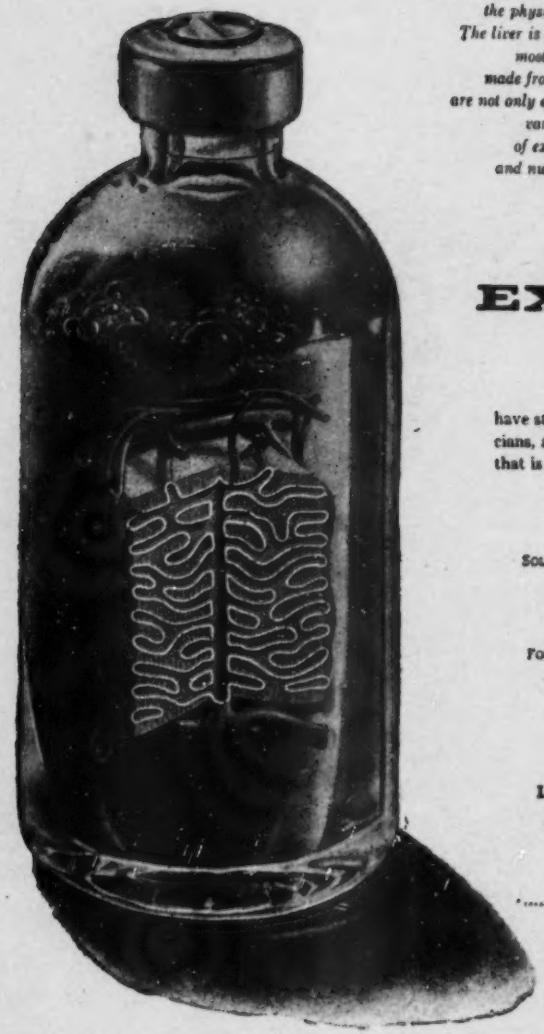

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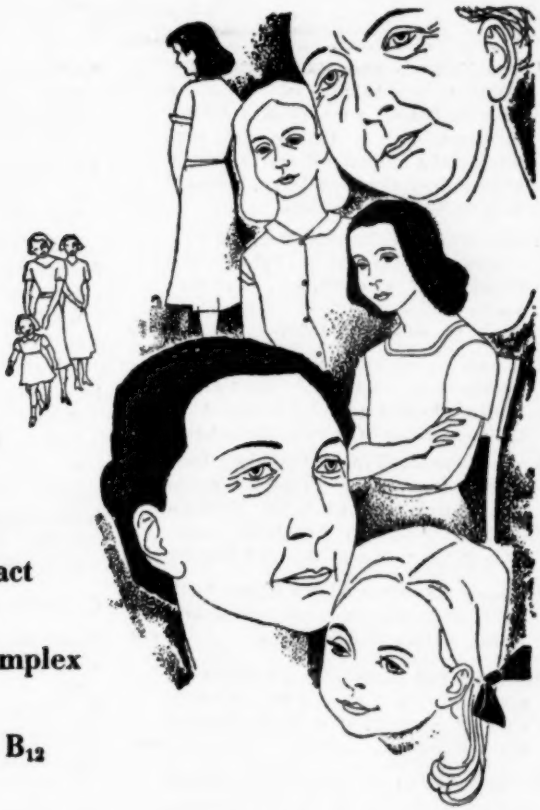

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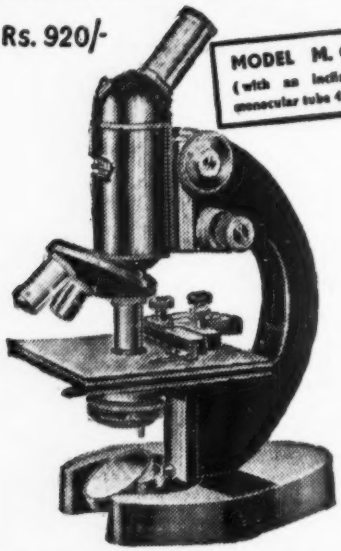

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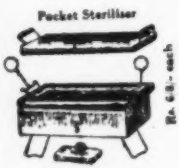

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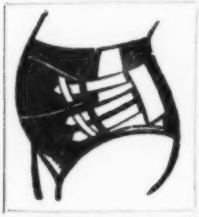

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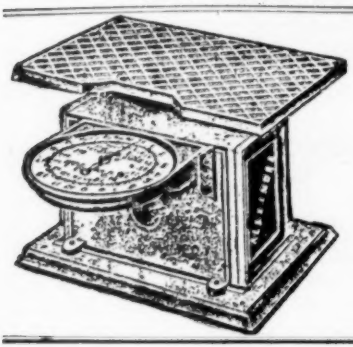

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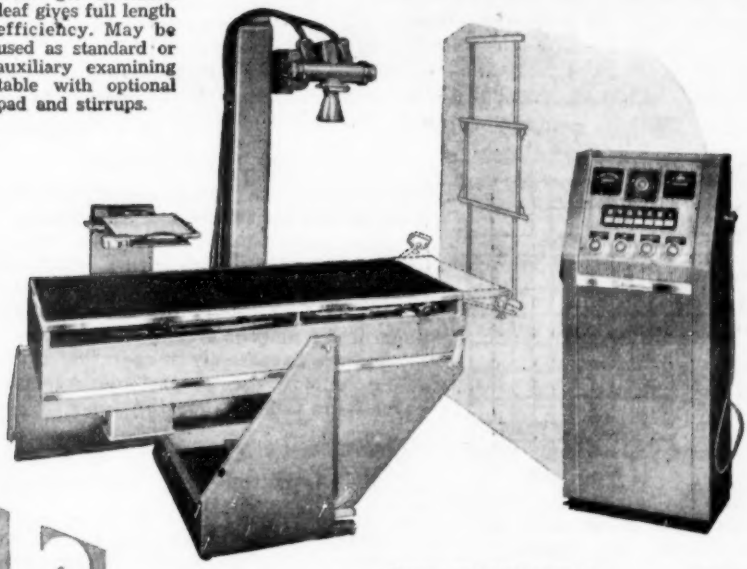

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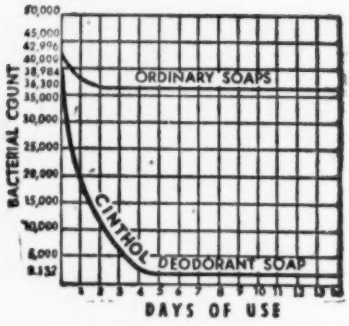

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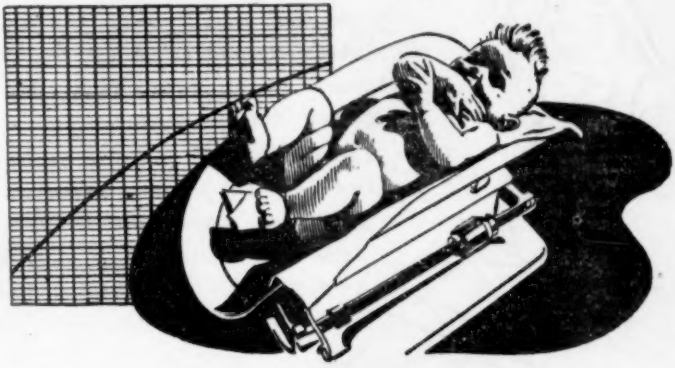

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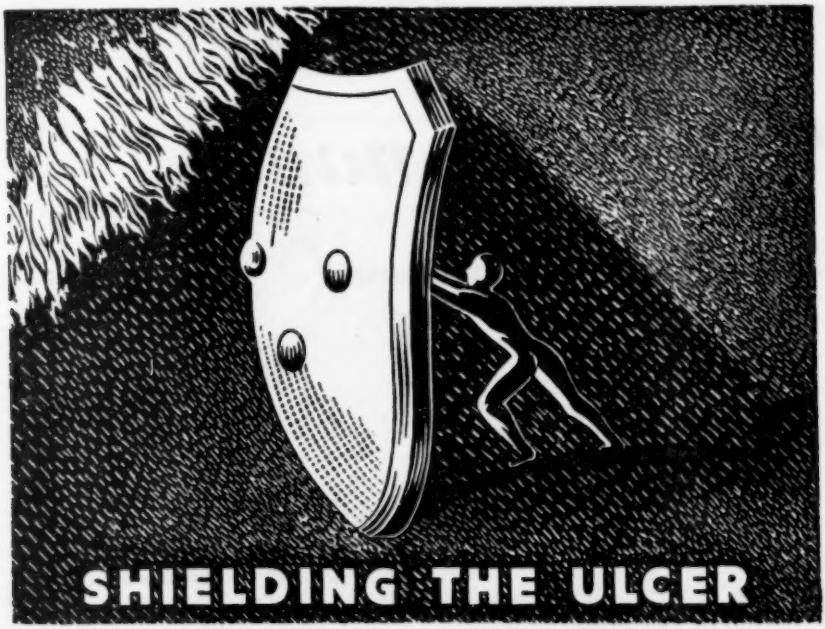

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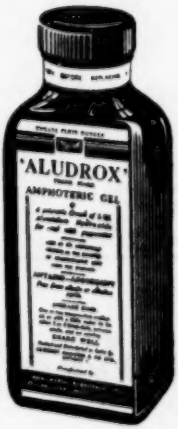

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Kuzell *et al*, *J.A.M.A.*, 149, 729, 1952.

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LUPUS VULGARIS AND ITS TREATMENT

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TUBERCULOSIS of the skin, though fortunately not a common disease and seldom a direct cause of death, has always been regarded in the past with ample justification as one of the most intractable and discouraging of maladies to deal with.

The cutaneous manifestations of tuberculosis are usually classified into two main groups :—

GROUP I.—Lesions which are of a proved tuberculous nature and in which tubercle bacilli may be demonstrated,

GROUP II.—Tuberculides—associated with a tuberculous focus elsewhere, due to the toxin or some breakdown products of the bacilli. It is an allergic phenomenon.

The clinical varieties commonly found here in India are lupus vulgaris, tuberculosis verrucosa cutis, scrofuloderma and lupus ulcer in the order of their incidence. They all belong to Group I. Lupus vulgaris is by far the most common variety, while tuberculosis verrucosa cutis is really a modified and indolent form of lupus vulgaris found as warty growth on the fixed skin of hand and foot, and buttock. The other group *i.e.*, tuberculide variety is rather rare in this country.

Mode of infection.—**A.** *Exogenous*:—(1) By inhalation through the mucous membrane of the respiratory tract, where it is deficient or diseased and then by lymphatic permeation or direct extension.

(2) By simple contact with infected sputum. Exogenous infection occurs particularly on the knees and buttocks in children due to their crawling habit.

B. *Endogenous*:—From some hidden tuberculous focus, organisms find their way into the blood or lymph stream when the resistance of the patient is lowered and are ultimately deposited in the skin.

AETIOLOGY:—(1) The essential ætiological factor is a local tuberculous infection. The tubercle bacillus though most difficult to find, is probably present in some stage of the disease and tuberculous infection can be produced by inoculation from the lesions.

The tubercle bacilli are rapidly destroyed in the skin, which explains why they are only to be found with difficulty.

(2) Heredity:—The disease is not hereditary—possibly a child may inherit a tissue susceptibility to the disease.

(3) Race:—Certain European races are particularly susceptible to the disease.

(4) Age:—Disease of the children—commonly starts in their childhood.

(5) Sex:—More readily affects females than males.

(6) Class of people:—Ill fed, ill-housed, ill-nourished and ill-clad. Lack of education very often results in the disease being concealed until it has reached an advanced stage.

(7) Determining factor:—Acute fevers, *e.g.*, measles, appear to be the determining factor in the development of lupus.

(8) Pre-existing lesions or scars may be the starting point in a certain proportion of cases.

Pathological changes with symptomatology:—The nodules of lupus vulgaris arise in the lymphatics of the dermis. The blood supply of the affected area is rapidly cut off by a proliferative endarteritis. There is proliferation of epitheloid cells and an extravasation of small mononuclear cells. Giant cells of tuberculous type are formed and few tubercle bacilli may be demonstrated. The nodules go on spreading upward—epidermis becomes thinned out and loses its follicular character. It gradually comes to the surface of the skin as a pin-point nodule which is translucent and on diascopic examination assumes an 'apple jelly' colour whilst the surrounding skin is blanched by compression of the capillaries. The nodules are softer than the skin and several nodules may coalesce. Sometimes ulceration occurs or the nodules

may retrogress and finally disappear. The nodules are invariably replaced by white pliable scars.

In untreated cases more and more nodules are formed at the periphery of the diseased areas, whilst the oldest nodules in the centre retrogress and are replaced by scar tissue; the typical lupus vulgaris patch is thus formed and consists of:—(*a*) small nodules arranged peripherally; (*b*) larger nodules forming an intermediate layer; (*c*) whilst in the centre, is a white pliable scar.

Site of lesion:—Chiefly on the face but common also on buttocks, knees, elbows, fingers and toes. On the feet, hand and on the fixed skin they appear as warty growths. Lupus nodules may occur on the nasal mucous membrane and in the mouth. If the nose is invaded the cartilage of the septum may be eroded and the typical parrot-beak nose develops. The scalp is relatively immune to the disease; it is never seen on the genitalia.

Histopathology:—The lupus nodule is a granuloma consisting of masses of round cells, endothelial cells and lymphocytes and fibroblasts in a fine reticulum of connective tissue. Giant cells are found usually in the larger nodules along with plasma cells. Tubercle bacilli are rarely seen and if present are found in the giant cells or between the epitheloid cells. Small blood vessels are found in between lymphocytes and plasma cells. The centre of the lupus undergoes fatty degeneration but usually there is no caseation. The process destroys the glands and the hair follicles and the normal structure of the skin is finally replaced by scar tissue.

Complication.—(1) Deformity and disfigurement. (2) Chronic ulceration. (3) Epithelioma occasionally develops and lupus scar tissue—possibly due to prolonged treatment with irritant application of X-ray therapy.

DIAGNOSIS:—Most of the cases can be diagnosed clinically by the characteristic appearance. History of long duration, resistant to ordinary lines of treatment, absence of gross sepsis and constitutional symptoms, age incidence and site of election are helpful points for diagnosis. The presence of the lupus nodule and the characteristic 'apple jelly' appearance by diascopic examination are almost confirmatory. While in still doubtful cases histopathological study may become necessary for final diagnosis.

DIFFERENTIAL DIAGNOSIS:—The more important conditions to be considered and eliminated before the diagnosis of lupus vulgaris is settled are pyogenic granuloma, oriental sore, dermal leishmanoid, gumma, lupus erythematosus, rodent ulcer and epithelioma.

TREATMENT:—Before the introduction of any specific remedy, the treatment of lupus included measures to improve resistance of the patient by good food, general hygienic measures and by administration of tonics and vitamins. Dietetic treatment received much attention.

Nutritional therapy of lupus became popular at one time and hundreds of patients were treated in various clinics demonstrating that the skin lesions can be healed completely by the nutritional regime. The diet provided 1·5 g. protein, 2·7 g. fat and 4 g. carbohydrate per kg. of body-weight excluding common salts and the foods containing common salts. The foods were specially selected to safeguard a high content of vitamins, enzymes and minerals. The regime is supported by phosphorised or plain cod liver oil and a calcium mixture. The underlying principles of the therapy involve the whole field of blood chemistry and a change in the soil in which the tubercle bacillus grows.

Tuberculin injections have also been used until recently in graded doses over a long period. So long as over-treatment can be avoided, good results may be achieved. The ulcerative and hypertrophic varieties do not respond in general so well as the more atrophic types.

Cod liver oil was regularly prescribed in the nineteenth century, to every type of tuberculous case. In the year 1840, Emery, a French physician from St. Louis Hospital (Paris) reported a high proportion of cures in patients with lupus with 20 to 40 ounces of cod liver oil daily. Emery was successful in this manner and cured some cases. Nevertheless the treatment failed because of the poor tolerance of the patient.

In the latter part of the nineteenth century local treatment by concentrated *ultra violet therapy* was introduced by Finsen and it was further improved by combining it with general irradiation of the whole body by carbon-arc lamp baths. Many complete cures were effected by this method. But as it required much experience and skill on the one hand to carry out and the duration of treatment was too prolonged with very slow progress, many patients could not stick to the treatment till completely cured.

Calciferol has been used extensively—following the publication in December 1943, of Charpy's original observation. During the war year when Charpy's work was prosecuted, Dowling and Thomas in England independently treated cutaneous tuberculosis with calciferol. They reported (1946) approximately the same good results that Charpy had achieved. Later, calciferol has been used extensively in different countries by workers who got similar results.

Calciferol is given as high potency ostelin tablets each containing 50,000 I.U. or as a solution in alcohol each ampoule containing 500,000–600,000 I.U. or parenterally in oily solution.

Doses recommended are: for children–100,000 I.U. daily and for adults—150,000 I.U. daily or 600,000 I.U. I.M. biweekly. In the absence of toxic effects, it is continued for one month and then the dose is reduced. Treatment should be continued for a few months after all the signs and symptoms of the disease have disappeared

and the patient should be kept under observation for several years.

Charpy (1948) observed that vitamin D is more active when taken by mouth, because of better absorption. In general, the alcoholic solution seems to be more effective than the oily solution.

Dowling, Gauvain and Macrae (1948) observed in a series of 84 cases that response to treatment by injection was notably slower than by mouth but on the other hand, no toxic effect was encountered.

At the skin department of the Calcutta School of Tropical Medicine, considerable number of cases have been treated with calciferol since 1947. The doses of calciferol used generally were 500 to 600,000 I.U. I.M. biweekly for 2 to 3 months and then weekly for subsequent months with good results. There was no toxic reaction in any case that received injection treatment, while a few children developed toxic symptoms by oral treatment with the alcoholic solution. Panja and Banerjee (1952) reported 30 cases of cutaneous tuberculosis treated with calciferol in doses of 500,000 I.U. I.M. every other day for 3 weeks and then at 2 days' interval for one or two months with good result and without any toxic complication.

The mechanism by which calciferol exerts its beneficial effect is yet unknown. It is thought that calcium metabolism and mobilization are concerned in some way.

Charpy (1947) expressed the belief that calciferol reacts on the lupus not by itself but by phosphatase transferred to the skin by means of the circulation.

Contra-indications for calciferol therapy are:—Chronic kidney disease with albuminuria, pulmonary tuberculosis and uncompensated heart lesions.

Streptomycin, either alone or in combination with calciferol has been used with very good results. The usual dose of streptomycin is 0·5 gm. I.M. 12 hourly for a month or so.

Cornbleet (1948), Mackenna and Russel (1949) and others reported that cases which failed to respond to calciferol treatment alone, were cured completely by streptomycin.

At the Calcutta School of Tropical Medicine better results were obtained with combined streptomycin and calciferol treatment than with either of the two used alone.

Indiscriminate use should not be made of streptomycin as there is a chance of development of resistant-organisms and toxic reactions.

Para-amino-salicylic acid (PAS) has been demonstrated by Lehman (1946) to possess a bacteriostatic activity against the

tubercle bacillus in man, and this has been amply confirmed by other workers.

There have been a few reported cases of lupus vulgaris treated with PAS alone but it seems that the value of the drug is more in delaying and in suppressing the development of the resistant strains of tubercle bacilli when used in combination with streptomycin or the recently introduced Isoniazid. The dosages used are 8 to 12 gm. daily, divided into 4 to 5 doses by mouth.

Local infiltration or dressing with 10% Na PAS has also been tried.

Isonicotinic acid hydrazide is the latest chemotherapeutic agent used in different forms of tuberculosis. In cutaneous tuberculosis it has given very encouraging results, and already some reports are available about the efficacy of the drug. Dowling *et al* (1953), Russel and Thorne (1953) and others reported excellent results with isoniazid. Chakraborty *et al* (1953) obtained striking improvement with isoniazid in cutaneous tuberculosis and reported their results of treatment in a series of 36 cases. 21 cases were cured completely and 13 cases showed marked improvement, 2 cases became resistant to the drug after showing considerable improvement. These cases were subsequently treated successfully with calciferol.

The doses of isonicotinic acid hydrazide used is 100–200 mg. daily (adult) in 3 or 4 divided doses orally for 2 months or more. The incidence of toxicity to isoniazid appears to be low. Some of the common side-effects met with were:—giddiness, constipation, nausea, vomiting, thirst, retention of urine. Long continued treatment may result in damage to the internal organs like the kidney and liver. It may be concluded that isoniazid is a very effective drug in cutaneous tuberculosis. It is cheap, easy to administer and without notable toxicity.

Local therapy:—Complete removal of early lesions by surgical excision and local destruction of the diseased tissue by curettage or by cauterization has been aimed at, in the past. Local ultraviolet therapy by Finsen and Kromayer lamp once popularly adopted is gradually becoming out of date since the advent of successful chemotherapeutic agents in the field of treatment.

Summary.—(1) General features of lupus vulgaris have been described. (2) Treatment of lupus, both past and present has been outlined. (3) Recent treatment with calciferol and streptomycin has changed the prognosis of the hitherto intractable and discouraging malady. (4) Isoniazid, the latest chemotherapeutic agent against tuberculosis, has been found to be highly effective in lupus. The drug is cheap, easy to administer and without notable toxicity. It needs however, further trials and observation.

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Treatment of Lupus

The general practice in Europe in treating lupus, according to a Spanish physician, is to administer doses of six hundred thousand units of vitamins at short intervals to start with and then increase the intervals over some months. But the patients have to be kept under very careful and strict watching in order to prevent possible ill-effects of a serious nature. The standard procedure in the United States, favoured by nearly all dermatologists tends towards the combined use of calciferol and dihydrostreptomycin. The usual dose of calciferol is 150,000 units per day and that of dihydrostreptomycin is 1 gm. Undesirable side-effects should be carefully watched and suitably dealt with.—(*Jour. Amer. Med. Assoc.*, 146 : 299, 19-5-1952).

Pyogenic Infections Treated with Neomycin

Neomycin is active against a wide range of bacteria. Tests *in vitro* show that it is superior to other antibiotics against hæmolytic Staphylococcus aureus and albus, pseudomonas and proteus. Bacitracin, aureomycin, terramycin and chloramphenicol, however, are more active than neomycin against the hæmolytic streptococci. Neomycin is best used for topical application as an ointment in the strength of 5 mg. per g. of a soft paraffin and lanolin base or as an aqueous solution containing 1 to 5 mg. of neomycin sulphate per c.c. Of 95 patients with impetigo, impetiginized dermatitis, ecthyma paronychia, or furunculosis, treated by Livingood *et al*, 88 responded well to these topical applications. The results in otitis externa were particularly very good. Prolonged application of neomycin produced localized cutaneous moniliasis in seven out of the 95 patients.—(*J. Am. Med. Assoc.*, 148, 2.2-1952, pp. 334-339, *Abst. World Med.*, 12 : 2, p. 633, 1952).

TREATMENT OF TUBERCULOSIS OF THE SKIN

OBSERVATIONS IN A TUBERCULOSIS CLINIC

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THE commonest forms of skin tuberculosis seen in a tuberculosis clinic are:—(1) Scrofuloderma. (2) Tuberculous ulcers. (3) Indolent B. C. G. ulcer. (4) Ulcers after tuberculin testing in mass campaigns. (Not exactly tuberculosis of the skin). (5) Rarely lupus vulgaris:—(Usually these cases of lupus go to the General Hospitals and that is why they are only occasionally seen at the tuberculosis clinic).

Scrofuloderma.—It is also called "tuberculosis colliquativa", scrofulous gummata, or tuberculosis fungosa cutis, depending upon the physical appearances of the lesion.

It is a deep chronic involvement of the skin by extension from some structures beneath the skin, *i.e.*, commonly caseating tubercular glands, occasionally tubercular joints and bones. The commonest age periods are childhood and adolescence; and the commonest site is the neck region; face and axillary regions coming next.

The disease begins as firm subcutaneous nodules usually over the caseating tubercular glands in the neck; these nodules gradually enlarge and become attached to the skin, which turns red. After a few weeks to a few months, an abscess forms and breaks perforating the skin in one or more places or entirely destroying it and leaving an ulcer which is irregular with overhanging edges and uneven floor, sometimes communicating by a sinus with a deeper caseating gland.

There is a marked tendency to the formation of hypertrophic tuberculous granulation tissue often hiding the mouth of the sinus, and forming bridges of granulation and scar tissues. Pain is usually very little. There is often a seropurulent discharge.

After months or years, when healing has taken place, it leaves a characteristic uneven contractile scar frequently with bridges of scar tissue.

Prognosis depends upon the general condition and the nature of the treatment given. Without specific treatment the local healing takes place with disfiguring scars as outlined above.

TREATMENT:—(1) General. (2) Combination of antimicrobial drugs. (3) Ultra-violet light therapy. (4) Surgical dissection of deep caseating or large glands.

1. *General*:—(*a*) Children should be removed from proximity to and contact with the open cases of pulmonary tuberculosis to clean, open and healthy surroundings. Our experience of

hundreds of families in Delhi shows that the removal of the child to healthier surroundings is not feasible in actual practice owing to the present conditions of economic and housing distress. In the majority of cases all that has been possible to achieve is the isolation of open cases in a separate room (if more than one room is available in the house) and the proper disposal of sputum.

(*b*) *Diet*:—The diet should consist of plenty of milk, curds, cheese, eggs, fruits and green vegetables. An effort should be made to reduce salt in the diet.

(*c*) *Rest*:—The child should be prevented from activities involving physical strain and should be encouraged to keep busy in restful occupations and recreations.

2. *Ultra-violet light exposures*:—Our experience even prior to the days of antimicrobial drugs showed very satisfactory results with this form of treatment, both for skin tuberculosis as well as for tubercular glands. Regarding the dosage we use the Alpine Lamp at 36" distance. Exposures are begun with 1 minute given on alternate days and increased by one minute on every third exposure. If reactions like a rise in temperature etc., occur we miss out the next exposure and do not increase the time quickly. When the exposure time has reached 15 minutes, we continue it for another 10 to 15 exposures, and watch the results. If necessary we give two to three such courses. Ultra-violet light therapy is not given when there are symptoms of marked or moderate toxæmia or an associated lung lesion.

3. *Antimicrobial drugs*:—It has now been established for over an year and a half that it is inadvisable to use any single drug but to use them only in combination, in order to delay the appearance of resistant strains. Formerly when the use of a single antibiotic or chemotherapeutic agent was in vogue, we did not use them in cases of tubercular glands with or without scrofuloderma unless there was either a sinus or manifestations of toxæmia. The reason for such hesitation was again the fear of the appearance of resistant-strains. But since it has now been shown by various workers that when dihydrostreptomycin is used in combination with PAS, or Isoniazid the appearance of resistant strains is considerably delayed, we have started giving the benefit of this combination of anti-tubercular drugs to the patients with gland and skin tuberculosis in order to assist their powers of resistance and thus materially hasten recovery.

Dihydrostreptomycin intramuscularly twice a week (about 20 mg. per kg. per dose) along with Isoniazid (3 per kg. per 24 hours in 3 divided doses) should be given. This combination is given for a period of atleast two to three months.

4. *Surgery*:—All deep foci of caseating glands discharging through a sinus and very large glands should be dissected out under

the protection of antibiotics. Such a procedure undoubtedly hastens healing, improves the scars, and minimises the chances of relapse.

We combine all the above forms of treatment, surgery being done only when there is a definite indication for it.

Occasionally cases of tubercular ulceration of the skin also present themselves for treatment. Since most of them have super-imposed secondary infection also, they are given an ointment containing 5% PAS and Cibazol. Once the secondary infection is eradicated, they receive PAS ointment only.

In addition they also receive all the treatment outlined above (except surgery).

Recently two cases of indolent B.C.G. ulcer, were observed. Since more than six weeks had elapsed (which is the average time for the development of tubercular allergy) they were advised 5% PAS ointment and ultra-violet exposures. The ulcer healed with this treatment all right.

During the B.C.G. campaign in Delhi State, a few cases of deep ulceration after the tuberculin tests, came for advice. Such deep ulceration was probably due to secondary infection of the blister *plus* perhaps an added factor of high allergy with destructive response. The lungs of these cases were found clear on screening and there were no other clinical symptoms of activity.

These ulcers healed under treatment by washing with boric lotion followed by the application of Cibazol ointment. But one case, in which a leucoplast was used over the Cibazol ointment, developed a patch of severe allergic dermatitis round the ulcer. The outline of the area of allergic dermatitis was similar to the area of the leucoplast applied. Therefore, it was fairly obvious that the leucoplast had made matters worse.

In this case Cibazol ointment was substituted by Phenergan ointment (antihistamine ointment) and Phenergan tablets by mouth; the ulcer healed in a week's time and the dermatitis abated; but when the antihistamine drug was stopped itching reappeared in the patch of dermatitis and the area became red. Eventually the whole thing healed with Nixoderm ointment.

Rarely a case of lupus vulgaris comes in. In a recent case healing took place in 4 to 6 weeks' time under ultra-violet light therapy and 5% PAS ointment. He is still receiving the light therapy. I am reminded of another case of lupus vulgaris treated in a sister institution by topical application of Isoniazid in the strength 1 : 1,000. The ulcer healed and remained well for 3 months after cessation of treatment, *when relapse occurred.* Topical application of Isoniazid has been restarted and the results are being watched.

The modern treatment of skin tuberculosis may be summed up as follows :—

1. General treatment as outlined above. 2. Dihydrostreptomycin 1 gm. twice a week and Isoniazid 50 mg. three to four times a day (depending on the weight of the patient) for about three months. 3. Exposures to ultra-violet light therapy or if it is not available, the administration of vitamin D_2 about 1 lac units daily for at least three months. 4. Topical application of a 5% PAS ointment. 5. In order to prevent relapse it is useful to give after two months, another course of vitamin D_2 (or U.V.L. treatment).

Skin Patterns of Allergy to Penicillin

Sensitization to penicillin may occur either from parenteral administration or from absorption into the mucous membrane of the oral cavity, vagina or rectum. It may produce on the mucous membrane a localized contact type of dermatitis or by absorption from the surface, a systemic reaction resulting in the appearance of one of the cutaneous eruptions, produced by penicillin sensitivity :—1. *Topical application* may cause (*a*) contact reaction of mucous membrane, (*b*) contact reaction of skin. 2. Systemic administration may induce (*a*) urticarial, including serum sickness type of reaction, (*b*) erythematous and erythematovesicular, (*c*) purpuric, (*d*) exfoliative, (*e*) erythema multiformæ, (*f*) papular urticarial, (*g*) bullous, with or without hæmorrhage and (*h*) fixed eruption. 3. Secondary to penicillin administration, there may appear (*a*) moniliasis and (*b*) black hairy tongue.

Much clinical trial has failed to produce any dependable skin reaction with which to predict susceptibility to systemic reactions. Caution should therefore, be exercised in administering penicillin to any patient, who gives a history of allergic manifestations such as severe asthma, hay fever, or cutaneous eruptions of questionable origin, particularly urticaria. The history of reaction to serum or vaccine injections in the past, is also very important in taking decisions about penicillin therapy. Most certainly a marked increase in cutaneous eruptions following penicillin usage, has occurred since the advent of the oil-wax and other delayed-absorption bases. Penicillin is administered every 3 to 4 hours in aqueous solution and there is therefore, the possibility that this return to the intermittent form of administration may account for its reported low sensitizing potential. There are undoubtedly certain persons who react to the vehicle alone and these would benefit by the use of the rapidly absorbed aqueous solution of penicillin.

TREATMENT :—Antihistamine therapy in rather large doses has been helpful in the less severe penicillin eruptions but has generally been disappointing in the serum sickness type of urticarial reactions—who need help the most. Procaine intravenously and adrenaline subcutaneously give only temporary relief ; moreover many contra-indications to the use of adrenaline exist and its efficiency is lost long before the allergic reaction has begun to abate. Garret and Smith found that where no contraindications exist, ACTH or Cortisone (either the injectable or the tablet form of the latter) constitutes the best form of treatment.

They suggest that penicillin be administered *only* in those cases in which the welfare of the patient necessitates its use. Pustular dermatoses, furuncles which could be treated surgically, and mild febrile disorders of unknown origin are conditions for which *penicillin is frequently given* and in which penicillin therapy *is not absolutely necessary* due to the availability of other equally effective medications, far less likely to produce sensitization and allergic reactions.—(*Southwestern Medicine*, Nov. 1952).

DERMATOLOGICAL AND IMMUNO-BIOLOGICAL PHENOMENON

IN FUNGAL INFECTIONS

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FUNGUS infections are of such common occurrence that it is necessary to consider mycotic disease in the differential diagnosis of practically every obscure infection. Unfortunately very few are capable of making gross identifications of the molds, yeasts, and yeast-like micro-organisms encountered in the lesions of patients, on their skin, or in exudates or sputum. The lesions of internal organs, lungs, liver or intestines are frequently diagnosed as mycotic only after long delays caused by prolonged "Elimination" procedures, often impeded by a presumptive diagnosis of tuberculosis.

Actinomycosis, blastomycosis, coccidiodo mycosis, moniliasis, histoplasmosis, maduromycosis, aspergillosis and dermatomycoses—just to mention a few diseases of clinical interest, indicate how varied and widespread are the manifestations of fungal infection.

Fungal infections are so very prevalent that they should not be the subject of interest only to a few specialists but should receive close attention and study by all physicians. But unfortunately, in this country, medical mycology is only of esoteric interest.

There is a very close analogy and in many respects identity between the results of immunologic studies with fungi and those with the tubercle bacilli. Studies of cases with the fungal infections constitute simple and excellent means of investigating immunological happenings in the system—infections in general and tuberculosis in particular.

Paradoxically the pathogenic fungi have been the very beginning of the bacteriological era. The recognition of the genus Achorion as the cause of favus might be said to the first one in the identification of "micro-organisms as causal factors of human disease."

The immunologic experiments on fungi date as far back as 1902 when Plato and Neisser produced and used Trichophytin, analogous to Koch's tuberculin. Later came the masterly study of Gruby and Sabourard—the French dermatologists who classified the various types of fungi found in man and proved the specificity of fungal infections.

The manifold advantages of animal experiments with dermatophytic fungi soon become apparent. Animals were not killed by the disease, but was practically confined to the skin and its appendages thus making direct visual observation possible, the microscopic;

histopathological and immunological studies could be conducted easily at desired intervals. Elaborate laboratory precautions were not necessary and finally there was the definite predictability of the course of infection. With such ease in experimentation it is no wonder that many fundamental immunobiological principles of infection have been demonstrated with experimental fungal infections—Achorion quinckeanum in guinea-pigs at quite an early date, many preceding those of tuberculous infections.

A few of the salient inferences from the experimental findings are :—(1) The development of hypersensitivity to the fungus allergens–like trichophytin–is intimately connected with the appearance of inflammatory disease. (2) The degree of the hypersensitivity of the tissues is closely related to the severity of the manifestations, the time interval, tendency to healing and the number of fungi present.

The following may be postulated in applying these inferences to human disease :—

The disease due to these groups of fungi (hyphomycetes–dermatomycetes) are caused not by *toxins* but by *fungus allergens* similar to anaphylaxis, hay fever or eczematous dermatitis. These fungal diseases are allergic sensitisations. Moreover as shown by Bloch *et al*, one of the most important allergenic principles in these fungi is a polysaccharide and *not* a protein.

Fungal diseases are thus seen to be analogous to diseases like tuberculosis and leprosy whose micro-organisms also produce allergens which sensitize tissues and give rise to local allergic reactions which constitute the principal manifestation of the disease.

The clinical and theoretical significances of these facts take us far beyond the scope of the present discussion and so only a few points of practical importance will be stressed :—

The allergic infections of diseases are best treated by desensitisation or hyposensitisation measures like hay-fever-desensitisation with the specific pollen allergens. But unfortunately the method is therapeutically not a uniform success because of many unpredictabilities. Sensitivity may in certain cases, be enhanced instead of being desensitised. Here again we find a marked resemblance to "Tuberculin desensitisation".

This naturally leads to the much discussed controversial question whether the hypersensitivity to allergens of micro-organisms (Trichophytin or tuberculin) is protective or harmful ; is it a manifestation of immunity or an expression of the disease ? No immune reaction is known to produce increased inflammatory response. On the contrary, a reaction due wholly to immunity should be characterised by the reduction of the inflammatory response. Thus, we find the allergic reaction is unequivocally and obviously harmful. But at the same time the resulting tissue-reactions confer many

forms of beneficial and protective factors which indirectly help in localising the disease.

The higher the trichophytin sensitivity (*Cf.* tuberculin) the greater is the tendency to spontaneous healing, which readily explains the difficulties in controlling an epidemic of scalp infections in children with anthropophilic microsporon audouini in contrast to the more inflammatory but readily curable microsporon canis.

Epstein's experiments show a zone of local specific immunity at the centre of a ringworm patch and also at the periphery of the enlarging lesion, which account for the configuration of the skin patches, pathogenesis and immunology of trichophytids; the relationship of fungus hypersensitivity to anaphylaxis and the study of allied problems have provided a wealth of immunological data which have been utilised in elucidating many obscure immunological problems connected with other infections in man, like tuberculosis.

Summary.—Fungal infections are of very common occurrence and the mycotic diseases should be considered in the differential diagnosis of practically every obscure infection. The varied types and the widespread nature of fungal infections are mentioned. Fungal infections are simple excellent means for investigating immunologic phenomena. Many fundamental immunological phenomena have been demonstrated with experimental fungal infections. Attention is drawn to the close analogy of fungal infections to diseases like tuberculosis and leprosy.

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Therapy for Tinea for Capitis

Although X-ray epilation is the most efficient therapeutic measure, topical applications and techniques have been suggested during recent years.

In all inflammatory types of tinea capitis, and even some of the resistant, scaling variants, local measures should be given a fair trial for several months. The scalp should be shampooed twice a day and thoroughly anointed with a fungicidal ointment or solution. Too vigorous scrubbing should be avoided. Some of the most effective remedies include, salicylanilide (5 per cent), mixtures of the unsaturated fatty acids (propionic caprylic, undecylenic), iodine (1 to 5 per cent) thymol (1 to 5 per cent), ammoniated mercury (5 to 10 per cent) alone and in combination with salicylic acid (2 to 6 per cent), sulphur (5 to 10 per cent) and chlorhydroxyquinoline ointment. Dr. Howard T. Behrman, Dermatologist of the Mount Sinai Hospital (U.S.A.) has seen many localized patches cleaned out by means of careful and diligent measures of this type There have also been several recent and optimistic reports on the use of Asterol hydrochloride in tinea capitis. But Behrman considers that these reports need further confirmation.—(*The Scalp in Health and Disease* by H. T. Behrman, 1952, pp. 322-23).

SOME COMMON TYPES OF ALLERGIC DERMATITIS MET WITH IN PRIVATE PRACTICE AND THEIR TREATMENT

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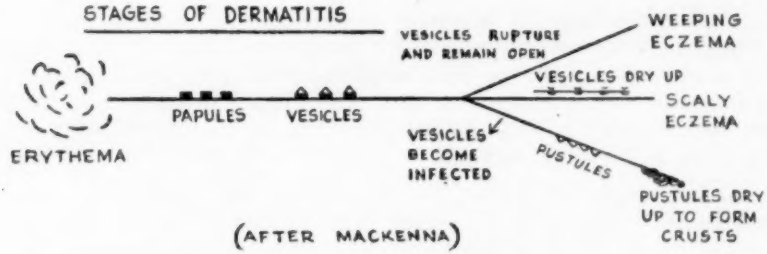
DERMATITIS means of inflammation of the skin and it is characterised by redness, swelling, heat and itching sensation with pain. The earliest sign of dermatitis is therefore, erythema accompanied by a little œdema. This is followed by formation of vesicles, or bullæ, which may later on become pustular, or might rupture, to exude serum which dries up to form crusts. The whole process might resolve with formation of a horny layer under the crusts, or relapse with further exudation of serum. The imperfectly formed granular and horny layers may peel off as scales (scaly dermatitis).



By the term allergy is meant sensitivity of the human system to a foreign body, either in the nature of a drug or a chemical taken internally or applied externally over the skin or the mucous surface. Hence by allergic dermatitis, we understand a condition caused by a particular foreign body (Allergen) on the system of a susceptible person, so as to bring about a cutaneous inflammation, the nature of which has just been described.

A few chemicals and drugs are commonly noted to cause allergic dermatitis. Most private practitioners would have come across a large number cases of allergic dermatitis in their practice. Sometimes they are baffled at the way cases indignantly defy all treatment or the morbid courses they take or at the extremely reluctant way in which they slowly become better at the expense of great time and money.

A. *Chemicals, minerals and drugs causing dermatitis by their external use (Contact dermatitis)*:—(1) Pigments in paints and brushes. (2) Lubricating oils. (3) Amidol and hydroquinone in photographic solutions. (4) Chemicals used in the rubber and vulcanizing industries. (5) Dyes and chemicals used in the textile industry. (6) Nickel and chromium salts. (7) Turpentine and

bichromate solutions in French polish. (8) Antiseptic lotions. (9) Chemicals and dyes in lipsticks, nail-polish, anti-sweat lotions, etc. (10) Paraphynelene-diamine dye in *kumkum*. (11) *Chunam* (lime) and cement. (12) Chemicals used in making hair shampoos. (13) Flours used in bakery.

B. *Drugs causing allergic dermatitis when taken internally either orally or by injection* :—(1) Heavy metals like arsenic, mercury etc. (2) Sulphonamides. (3) Penicillin. (4) Bromides and iodides. (5) Phenolphthaline. (6) Hypnotics. (7) Animal serum. (8) The new antibiotics, like aureomycin and hormones, like ACTH.

Contact dermatitis.—Almost all cases of contact dermatitis come under the group of industrial dermatitis occurring in factory workers. Though I am not dealing with these in detail, a few words regarding the methods of preventing industrial dermatitis are necessary at this stage.

Cool conditions of working, cleanliness of workers and their clothing, prevention of unnecessary exposure to irritants and mechanical protection by gloves etc., application of protecting substances, like fats and waxes and using only the mildest of soaps for cleaning after work, might help in bringing down instances of contact dermatitis in factory workers.

A few types of industrial dermatitis are mentioned herein. Builders and masons might suffer from contact dermatitis due to constant handling of chunam and cement. In bakers, sensitisation to flour might occur or a rash may be caused by salt. Hair dressers may be sensitised to ingredients in hair shampoos. Mechanics may become sensitised to ingredients in lubricating oils. Painters and photographers may develop dermatitis, the former due to drying oils or pigments likes zinc chromate and lead and the latter due to chemicals like hydroquinone, amidol etc.

Dermatitis also occurs in people engaged in the rubber industry especially, the vulcanizing side. Sensitisation by dyes and chemicals in garments might produce contact-dermatitis in people working in the textile industry. Nickel-platers and chromium-platers develop dermatitis due to the nickel and chromium salts. French polishers develop dermatitis due to turpentine and bichromates. Surgeons and nurses may develop dermatitis due to handling lotions and dentists might suffer from procaine sensitisation.

Dermatitis has been noted in modern fashionable women who use lipsticks, nail-polishes, anti-sweat lotions and depilatories etc.

Hindu women sometimes are allergic to *kumkum* containing the dye paraphenylenediamine. It is always therefore, advisable to apply *kumkum* over a thin layer of either eucerinum or vaseline previously smeared over the spot on the forehead. The same

dye is used to dye grey hairs and this might produce characteristic erythematous, vesicular dermatitis over the sides of the neck and the scalp.

Treatment of contact-dermatitis.—Usually contact dermatitis is localised to such parts as are directly in contact with the irritants, rarely a contact dermatitis manifests itself as a generalised exfoliative dermatitis. It is better to stop further contact with irritants and avoid such contact in the future. Contact dermatitis if localised will usually be erythematous or vesicular and rarely pustular in nature.

Early in the morning, one should apply 1% aqueous solution of gentian violet and then follow it by a liberal application of cremor zinci (G. H. Pharmocopœia) over the paint, which can be repeated later two or three times in the course of the day, or a well diluted solution of liquor plumbi subacetatis may be allowed to drip over pieces of gauze dipped in the same solution and kept over the affected parts for about 2 hours every day. If the dermatitis affects the feet or the hands they can very well be dipped in warm weak boric baths *i.e.* 2 spoonfuls of boric acid diluted in a basin of warm water.

It is better to avoid sulphonamide dressings externally and penicillin ointments are useless. Aqueous penicillin may very well be given parenterally and as a local spray (dilution 1 cc. 1000 units). It can be sprayed several times during the course of a day.

Antihistaminic drugs have very little value internally or externally in skin allergy. Probably they may themselves start a reaction. Even if there is some relief, it will be only of temporary duration.

The administration of vitamin C is essentially indicated. Massive doses can be given by mouth as well as by injection through the vein mixed with calcium and glucose. Riboflavin may be given daily in doses of 10 to 20 mg.

When the dermatitis has improved *i.e.* when the condition has stopped oozing and there is diminished itching it is better to strengthen the external applications by adding a little ichthyol or coal-tar which stimulates the formation of fresh skin while at the same time lessening the itching. Later on, stronger applications containing acid salicylic preferably in the nature of a paste (Lassar's) may be used to remove keratinisation.

At this stage the patient may be desensitized to the particular allergen by non-specific protein therapy. The patients' own blood may be injected into their muscles at regular intervals in increasing doses. This is termed auto-hæmotherapy. Usually one can start with $^1/_2$ cc., increasing by $\frac{1}{2}$ cc. each time and end up with 3 cc. of blood given once in 4 days depending on the severity of the

reaction in the patient. Sterile milk may also be given in the same way. T.A.B. vaccine may be given intravenously starting with 10 millions in young and robust individuals. Parke Davis "Lertigon" may be given starting with 0·01 cc., gradually increasing by 0·01 cc. each time until a final dose of 0·08 cc. is reached. It should be well diluted with distilled water. It is an antihistamine protein complex and has acted well in a large number of cases. Simultaneously the general health of the patient should be built up with liver extract injections, Vitamins B_{12} and B-complex, iron, calcium, etc. Protein foods preferably those containing liver like Ledinac (Lederle) are very useful.

The same treatment may be carried out for dermatitis caused by drugs taken internally and absorbed by the system. But some of them require more elaborate specific antidotes.

Dermatitis caused by drugs taken internally—Arsenic, gold, bismuth and mercury.—These heavy metals are not so commonly used nowadays as they were a few years ago, thanks to the advent of the new antibiotics in the field of therapy. But nevertheless many cases of arsenical dermatitis are seen in those who had had arsenic for the treatment of syphilis either from a modern qualified physician or from native *vaids* who specialise in distributing certain oils which, when taken internally for a specified length of time with the usual diet restrictions about which the physician is very strict, is supposed to cure all diseases of the skin, including those of venereal origin. Such oils are commonly sold in Madras. Some ignorant patients who regularly take these oils burst out with severe exfoliative dermatitis, pustular and stinking. We have seen such cases and they resemble more the exfoliative type of arsenical dermatitis. It is a pity that these oils which have a ready and quick market in Madras have not yet been analysed to find out what they contain. We have to infer from the effects produced on the patients that it contains some form of crude arsenic and mercury.

Arsenical dermatitis is usually seen in persons 8 to 10 days after the injection of arsenicals. If they have been taking arsenic by mouth as Fowler's solution it takes a much longer time, some months before the dermatitis breaks out on the skin. Very rarely arsenical dermatitis is noted after the first injection. Most cases give a history of having taken too many injections of arsenic at very short intervals. Two cases on our record gave a history of having taken more than three injections of neo-arsphenamine in the course of a week. Another patient had taken 14 injections of neo-arsphenamine for syphilis and his was the most severe case of arsenical dermatitis we have seen. Most of the cases probably suffered more from arsenical poisoning than from allergy. It is a pity that we still continue to use arsenical therapy in the treatment of syphilis while oil-penicillin and other antibiotics have superseded the arsenicals.

Even in the treatment of conditions like tropical eosinophilia, it is better to give milder arsenic preparations like acetylarsan or carbarsone rather than N.A.B. or mapharside. All arsenic injections should be well spaced, not more than once a week and during each administration the patient should be asked whether he had any itching after the previous injection and if so arsenic should either be stopped or given in lesser strength and with great caution. The urine should always be examined for albumin and sugar and if albumin is present in the urine, arsenic should *not* be administered at all.

Arsenical dermatitis is usually exfoliative in nature, but it may be erythematous, urticarial, vesicular, herpetic, pustular, ulcerative and gangrenous. In some cases sloughs of skin separate leaving raw areas. Layers of skin continually peel off, as the patient gets better and in one case we have noted the skin having peeled off three times in the course of four months. There may be a rise of temperature and the patient is usually bed-ridden, with bodily pains and extreme exhaustion.

Such patients should be made comfortable at home, preferably on Dunlopilo beds with a constant attendant to look after their needs. The urine, temperature and pulse should be checked up periodically. Diet should be restricted to fluids preferably glucose-water, orange juice and barley-water. Vitamin C and riboflavin should be administered in large quantities.

BAL injections should be started as early as possible. BAL is dimercaprol or British Anti-Lewisite marketed by M/s. Boots Pure Drug Ltd., London in 2 c.c. ampoules. Peters and his colleagues showed that after exposure to arsenical blister gases, the enzyme system responsible for pyruvate oxidation is inhibited. BAL prevents this inhibition and also reverses it when once it has been established. BAL is administered intramuscularly, 4 injections in the course of the first 24 hours, then 3 injections during the next 24 hours and later reduced to one or two injections a day depending upon the response, for three or four days. Remarkable improvement in the patient's general condition is noted after the administration of BAL. Usually there are no toxic reactions but in some cases there may be nausea or a rise of temperature or a dysentery-like syndrome might arise.

Externally zinc cream or calamine lotion is applied and aqueous penicillin is given in doses of 2 lakhs twice a day for about a week to combat any side-reaction or resultant bronchitis or bronchopneumonia.

When the patient has improved, his diet still essentially vegetarian may be gradually increased ; milk, butter-milk and fruits may be added. A few calci-ostelin and B-complex injections may be given and still later a few liver extract and B_{12} injections.

Liver protein oral preparations like Lederle's Ledinac may supplement. The eyes and mouth should be cleaned daily.

When crusts have formed, a mild sponge bath should be given without much rubbing and olive oil applied over the skin. Always hyper-pigmentation is noted in arsenical dermatitis which takes a pretty long time to disappear and the patient is advised never to take arsenic for the rest of his life. Dermatitis due to gold therapy is much more severe and BAL is effective in its treatment. Gold is very rarely used nowadays except in the treatment of tuberculosis and leucoderma. [The same treatment holds good for dermatitis due to bismuth which is sometimes used in the treatment of syphilis.]

Sulphonamides and penicillin.—Sulpha drugs after continuous usage produce typical erythematous, macular, papular, vesicular or exfoliative dermatitis. Usually it appears after a week but in sensitive cases, dermatitis has been noted overnight after taking a few tablets of a sulpha drug or after one dressing with sulphonamide powder or ointment. Sulphonamide and sulphathiazole are more toxic than sulphadiazine or sulphamerazine. It is always a good principle not to drug a patient with too much of sulpha drugs. It is reported that a small percentage of cases become light-sensitive for some months. Cases of sulphonamide dermatitis may be treated with aqueous penicillin injections and vitamin C, riboflavin and calcium orally with the usual external applications as in the case of acute dermatitis.

Very rarely does aqueous penicillin produce allergy. But procaine or oil penicillin disagree with many people. Some cases of procaine dermatitis are very severe and the patient becomes sensitive even to aqueous penicillin. Hence he has to be treated with massive doses of vitamins C and K, calcium and glucose. As already stated antihistamine drugs have very little action on allergic dermatitis.

Bromides and iodides.—Bromides and iodides taken continuously for a long time produce eruptions on the body. Bromide eruptions are pustular in nature, resembling pustular acne without the comedos. Bromide eruptions particularly take a long time to disappear even after the cessation of the drug. The simultaneous administration of arsenic as Fowler's solution, is stated to reduce the incidence of bromide allergy. Iodides produce similar pustular eruptions. Vesicular and bullous eruptions might also occur. The treatment of both the conditions is by stopping the administration of the drug and hastening elimination by the administration of sodium chloride by mouth.

Phenolphthalein, a favourite ingredient in most commercial purgatives, causes a vesicular and eroded eruption on the genitalia and on mucous surfaces.

Hypnotics like barbiturates have been found to produce acute rashes after prolonged use. Aspirin also sometime produces small macular rashes on the body.

Serum eruptions appear between the seventh and twelfth day after an injection of animal serum *e.g.* antitetanus serum. The eruptions are erythematous and urticarial If the eruptions are severe, adrenaline injections should be given subcutaneously.

The newer antibiotics.—ACTH and Cortisone produce hyperpigmentation and the formation of acne comedos and seborrhœic dermatitis probably due to the androgenic activity of the ACTH. Aureomycin which is perhaps the most powerful antibiotic in use at present produces glossitis, cheilitis resembling perleche. It also produces in some cases vaginitis and pruritus. All these are probably caused by the action of aureomycin in inhibiting the growth of bacteria resulting in yeast-like organisms being stimulated to grow in large numbers. Aureomycin itself was noted to cause dermatitis both when taken orally and when applied externally.

I have attempted to give a description of most types of allergic dermatitis commonly met with in daily practice, so that it may prove useful to the general practitioners.

I am grateful to Dr. S. Rajagopalan, L.M.S., F.D.S., Hony. Dermatologist, Stanley Hospital, for his very valuable and helpful advice. I am obliged to the Dean of the Stanley Hospital and to the Director of Medical Services, Madras for their kind permission to publish this article.

Pruritus Vulvæ as Seen in Dermatological Practice

Lynch reports on his personal series of 112 patients in whom pruritus vulvæ was the primary complaint. There were no examples of lichen planus or seborrhœic dermatitis and only once was contact dermatitis a primary cause of the pruritus vulvæ, though it was often a complication. The sequence of involvement of vulva and perianal regions and the nature of involved lesions were correlated with age of onset and with atopic, infective and psychological factors.

In 15 patients with localized periclitorial involvement, psychological factors were particularly evident. Menstrual irregularities were common, but the significance of endocrine influences is doubtful. 15% of the cases showed disturbances in sugar metabolism, associated with redness, swelling and bacterial infection. Vaginitis and discharge or other pelvic disease was present in 40 per cent of the cases. By way of treatment, Lynch recommends sedation, control of contributory organic factors, intelligent topical therapy, limited use of X-rays, and *"family doctor"* type of superficial psychotherapy. He condemns injection, vulvectomy and neurosurgical procedures.—(Lynch, F. W., *J.A.M A.*, 150 : 14, 1952. Abst. by A. J. R. in *Br. Jour. Dermat.*, April 1953).

PYOGENIC INFECTIONS OF THE SKIN

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PYOGENIC infections of the skin are very common in general practice, and sometimes offer considerable difficulty in treatment.

I. Impetigo contagiosa.—This is the commonest infection of the skin in children, and is one of the commonest of skin diseases. It first appears as a scabbed septic sore on the face, hands or knees. From these sites, it spreads asymmetrically.

Various types of impetigo contagiosa are described :—(1) Bullous type :—Appears as slightly raised flaccid blebs. The base is inflamed. (2) Circinate type:—The blisters of the bullous type rupture, the centre dries up and there remains a pale fringe of the blister with some exudate. This is the circinate type. The differential diagnosis of this type is from tinea circinata, but the latter runs a sluggish course and may take atleast a week to attain a diameter of an inch or two, though this happens in a few days in impetigo. (3) Scaly variety or impetigo pityroides :—This is seen towards the end of an attack of ordinary impetigo, and different types of lesions may be seen in the same individual. They are however, very common in children suffering from chronic nasal catarrh in whom there was no attack of ordinary impetigo. This variety (scaly variety) which closely resembles seborrhoeic dermatitis is very resistant to local treatment. Impetigo pityroides is contagious and epidemics may occur.

TREATMENT :—If the lesion is extensive, it is always better to apply *starch poultices* at the beginning. This treatment softens the crusts and helps in their removal. The infectious exudate is also absorbed and development of fresh lesions is prevented.

Starch poultices are prepared thus :—4 teaspoonfuls of starch and one teaspoonful of powdered boracic acid are taken and cold water is added just sufficient to make a thick paste. Boiling water is now added and the mixture is stirred constantly till the diluted cream sets into a jelly. This forms one pint of starch paste. It is then cooled and can be applied on a piece of sterile cloth.

The poultices should be changed three times a day and should be continued till the exudation practically ceases. One per cent solution of gentian violet may be applied once a day along with starch poultices. The part should be washed once daily.

For moist lesions, cleansing with isotonic saline and a mild antiseptic like acriflavine is good. In the acute stage, ointments should be avoided as they keep the skin moist and susceptible to infection. For the face and other exposed parts, zinc calamine lotion with 0·1 per cent mercuric chloride is good.

When the skin becomes dry under treatment with a lotion, a zinc paste with 3 p.c. yellow oxide of mercury is used :—

Hydrarg oxidi flavi	...	gr. v to x
Pasta zinci comp. B.P. ad	...	℥ i

I have found that a paste consisting of 0·2 gm. of sulphanilamide powder, 0·2 mg. acriflavine, and 1 gm. acid boric in rose water applied to the affected area gives very good results. Patent pastes like Dermoquinol, Vioform cream and Sterosan paste have also given very good results. Ung. Hydrarg ammoniata 1% is also useful. A cream containing 500 to 1000 units per gm. of penicillin also gives very good results, though dermatologists rarely use this as well as the sulphanilamide cream, as they may irritate the skin and cause eczematous dermatitis. When the infection is severe and in intractable cases, penicillin by injection and sulphonamides orally are very useful.

II. Intertrigo:—Intertrigo affects the natural folds of the skin under the influence of moisture and friction.

Three varieties of intertrigo are met with :—(1) Pyogenic intertrigo which is a common sequel to impetigo. (2) Seborrhoeic intertrigo most commonly affects the retro-auricular folds in a patient with a scurfy scalp and with other areas of seborrhœic dermatitis. (3) Mycotic variety. It is rare in children.

TREATMENT :—For pyogenic intertrigo, the treatment is the same as for impetigo. The affected area may be painted with 1 p.c. iodine in spirit and a simple zinc paste then applied. This procedure should be carried out once or twice a day. The application of iodine in spirit is also very useful in the mycotic variety. The submammary, internatal and the retro-auricular regions are the usual sites. The parts should be washed twice or thrice a day with boric lotion, dried and talc powder then applied. The opposing surfaces should be kept apart by means of pads of boric lint. For the intertrigo affecting the back of the ear, an application of 2% solution of silver nitrate in nitrous ether is very effective. Patent preparations like dermoquinol, or sterosan creams are also useful.

Scrupulous cleanliness, mild antiseptic dusting powders, avoidance of overclothing, regular exercises in the open air, diet poor in fats and carbohydrates but rich in vitamins are all very useful. The internal administration of alkalies is also essential.

III. Furunculosis—(boils).—It is a deep staphylococcal infection of the hair follicles and sebaceous glands. It commences as a red, slightly raised area. This enlarges, hardens and becomes very tender. There may be considerable œdema all round. After a few days the apex becomes soft, pus comes out as also the necrosed tissue. The inflammation subsides and a scar remains.

The common sites for boils are the back of the neck, buttocks, back, thighs and forearms, though any part of the body may be affected.

TREATMENT:—The boil and the skin all round, should be painted daily with diluted Tinc. Iodine or 2% aqueous gentian violet, and a hot wet dressing of 10% ichthyol in water is applied every three hours. This is carried out till the boil bursts, the core of dead tissue comes out and the discharge has subsided. The boil can also be operated surgically. X-ray treatment is often useful. Avoidance of friction to the area is essential.

General treatment consists in examining carefully for a septic focus in the teeth, tonsils, nasal sinuses etc. Examination of urine and blood for sugar is essential, and treatmeat is then directed accordingly. I had a male patient, aged about 50 years, stout and apparently healthy who got recurrent attacks of boils. After some treatment locally, he got relief. He again got another attack, and a third one, and then came to me for advice. I found nothing wrong with him, and so I examined his urine for sugar. It contained 2·38% sugar.

In fatty, seborrhœic persons, restriction of carbohydrates and full doses of alkalies are indicated. In debilitated, anæmic persons in whom phosphaturia is common, iron should be given and also acid sodium phosphate to produce acidosis. Holiday in the open air, and sunbaths do good in obstinate cases. Injections of staphylococcal vaccine, injection of colossal manganese and tin preparations (Stannoxyl) orally are all useful. Auto-blood therapy is also effective. A course of penicillin injections is very useful. A liberal supply of vitamins should always be included in obstinate cases.

IV. Sycosis.—This is a pustular folliculitis of the hairy parts. It is a staphylococcal infection of the orifices of hair follicles occurring in areas where the hair is rough and well developed. Therefore, the commonest sites are the beard and moustache regions in men, but it can also occur in the eyebrows, axillæ, pubis and scalp.

The lesions are follicular pustules and papules pierced by hairs. They are usually confluent. The skin between the follicles is usually affected with diffuse staphylococcal dermatitis ("seborrhœic sycosis"). The infection in sycosis is superficial, and so the hair is, not as a rule, destroyed. This is just furunculosis in which the deeper layers of the skin are affected.

The characteristic feature of sycosis is the repetition of multiple superficial pustules in a localised area. This may persist for a long time—months or even years. Thus, infective dermatitis is associated with pustular eruptions and so in a fully developed case of sycosis, we see a combination of infective dermatitis and folliculitis.

TREATMENT:—The treatment of sycosis should be by the application of mild antiseptics after the part is shaved. But as the part

may be acutely inflamed, shaving will be very painful. So, during the first few days of treatment, the hairs are clipped and painted with gentian violet and starch poultices are applied. When inflammation subsides, crude tar may be applied. This is very useful. As a rule, ointments are not tolerated in sycosis, but ointments containing chlorohydroxyquinoline are useful. Patent preparations like Ung. Quinochlor Co. (Squibb), Dermoquinol, Vioform cream of Sterosan are all good.

Fractional doses of X-rays cause hair to fall and offer therefore, an effective remedy. Penicillin is applied in the form of liquid or as an ointment in the strength of 500–1000 units per gm. Parenteral penicillin is useless. Staphylococcal vaccines are not useful in this disease probably because the lesions are superficial.

Summary.—1. Impetigo contagiosa is the commonest skin disease in children. 2. Starch poultices are very useful in impetigo if the lesions are extensive. 3. Penicillin injections and sulphonamides orally should be used along with local treatment in intractable cases of impetigo. 4. Dermoquinol is useful for impetigo, intertrigo and sycosis. 5. Examination of urine for sugar is very important in recurrent attacks of boils. 6. Injections of collosol manganese and stannoxyl orally are useful aids in the treatment of furunculosis. 7. Sulphanilamide-acriflavine-acid boric in rose water is a good application for impetigo.

Aureomycin–Topical use in Skin Diseases

A three per cent aureomycin hydrochloride ointment in a petrolatum and wool-fat base was used by Solomons in the treatment of a variety of skin conditions. Thus, 22 cases of sycosis barbæ yielded well to this treatment, though some of these had previously resisted all other treatments. The ointment was applied thinly and gently to the involved areas twice a day and improvement occurred in one week. Only one of twelve cases of folliculitis proved resistant to this ointment. 55 out of 57 cases of impetigo responded well. The response was quite good in 12 cases of gravitational ulcer. Good results were also secured in 8 cases of post-auricular dermatitis. Solomons considers aureomycin ointment to be without question a valuable aid in treating pyogenic infections of the skin. Its local sensitizing powers are low and the resolution was exceptionally rapid in quite a large number of cases.

In other conditions treated by the author: no improvement occurred in 3 cases of chronic paronychia; impetiginized lesions in herpes zoster in 4 children were resolved in a week. Verruca vulgaris—warts disappeared in 3 of 12 patients after a month's treatment and verruca plana cleared in one patient in 4 weeks.—(*Br. Med. Jour.*, 1-9-1952).

BULLOUS ERUPTIONS

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BULLOUS eruptions are characterised by big transparent vesicular eruptions, containing a clear fluid, which affect the human cutaneous surface from head to foot. There is intolerable itching. On pressure, the vesicles rupture, and fluid exudes leaving dry crusts behind. It is auto-inoculable and the infection spreads throughout the body by scratching. Mucous membranes are seldom involved. Due to secondary infections, the clear contents of the vesicles become turbid and opaque due to pus formation. Bullous eruptions are of various sizes. They appear in any one of the following five skin diseases:—(I) Drug eruptions; (II) bullous urticaria; (III) bullous impetigo; (IV) dermatitis herpetiformis; (V) pemphigus.

I. Drug eruptions.—Due to individual idiosyncrasy, certain persons develop vesicular eruptions on the cutaneous surface after taking any one of the drugs: Potassium bromide, Potassium iodide, Chloral hydras etc. Such patients are susceptible to the particular drug all their life-time. Within an hour or two of taking it multiple bullous eruptions appear throughout the body with intense itching. In iodide rash, there is watering of the eyes and nose, besides puffiness of face, in addition to the vesicular eruptions. On stopping the offending drug, skin eruptions gradually subside within a day or two. In order to relieve itching and to hasten the drying of cutaneous eruptions, calamine lotion or cremor zinc may be dabbed externally. The drug that gives rise to these bullæ should be stopped at once.

II. Bullous urticaria.—Bullous eruptions due to urticaria are transitory in character. They appear on the skin-surface throughout the body and disappear automatically within a few hours, with or without treatment. It is an allergic reaction due to various causes, such as insect-bites, change of surroundings, change to plains or hill stations or hay fields as also personal idiosyncrasy due to drugs or diet. The cause has to be found and eliminated to clear up the eruptions. Multiple vesicular erythematous eruptions of various sizes appear throughout the body with intense itching.

TREATMENT:—Eliminate the cause of the skin eruptions to prevent further attacks. Externally, calamine lotion or any one of the antihistamine creams may be applied to relieve the itching and to hasten resolution. The market is flooded with antihistamine drugs, *e.g.*, Antistine, Anthisan, Syncopen, Benadryl, Avil, Trimeton etc. Urticarial rashes clear up within a couple of days by the administration of any of these preparations, either orally or by injection or both in severe intractable cases.

III. Bullous impetigo.—Bullous eruptions appear on the body due to streptococcal infection. The bullæ contain turbid fluid due to pus formation. It is highly infectious and is both auto- and hetero-inoculable. There is a pricking sensation, itching and hectic temperature in the evenings due to sepsis. These vesicles spread by scratching and pus oozes out on the surface, leaving behind stuck-on-scabs on the affected parts.

TREATMENT:—*Prophylactic*:—The infected cases should be isolated to prevent the spread of infection to other members in the house or hostel or school. Sterilise all used beddings and clothes. *Curative*: (*a*) Externally dab the eruptions with a colloidal suspension of Cibazol in calamine lotion, as it dries up quickly forming a coating on the affected parts. The action is efficient and the lotion penetrates quickly into the vesicles. It does not stain clothes as it dries up rapidly. This application is superior to the applications of Hydrarg. ammoniata or sulphonamide in an ointment base. (*b*) supplement the external medication with 2 injections of penicillin (2 to 5 lacs units) to eradicate the infection. (*c*) if the vesicles still persist inspite of the above treatment, give orally 2 to 4 capsules of aureomycin (250 mg.) one capsule after food, twice a day.

IV. Dermatitis herpetiformis.—It is characterised by multiple vesicular eruptions along the course of the peripheral nerves throughout the body. The vesicles are small, symmetrical on the body unlike herpes zoster, which is asymmetrical and stops at the middle line of the body. The eruptions are of various sizes and contain clear, sterile fluid characterised by intolerable itching and neuralgic pain. Itching is relieved only on breaking the vesicles by scratching. It is rare in the tropics. The ætiology is not known but is believed to be due to a filter-passing virus. Daily fresh crops of vesicles may appear on the cutaneous surface and rupture on pressure. Mucous membranes are seldom involved. The prognosis is poor.

TREATMENT:—Apply externally calamine lotion or zinc cream to dry up the serous exudations that emanate from the vesicles. Germanin (Bayer 205) which is a trivalent antimony preparation is injected intramuscularly to clear up the lesions. A course of six injections will suffice.

V. Pemphigus.—Occurs both in children and in adults. Usually persons of high social status are affected. It is characterised by multiple vesicular eruptions on the skin surface with a clear, serous, sterile fluid. Fresh crops of vesicles appear daily and old ones rupture by pressure. There is intense itching, not however, so marked as in dermatitis herpetiformis. It is caused by a filter-passing virus and is auto-inoculable. There are two types of pemphigus, *viz.*, pemphigus neonatorum and pemphigus vulgaris, which is however, rare in the tropics.

Pemphigus neonatorum:—Occurs in infants. The prognosis is very good and cent per cent recovery takes place. Usually the vesicular eruptions clear up within a week by external applications of calamine lotion or cremor zinc. It is not infectious and so there is no need to isolate affected children in the house.

Pemphigus vulgaris usually occurs in adults and may be acute or chronic. The prognosis is poor in the acute type, whereas in the chronic type, prognosis is fair. The bullæ contain a clear, sterile serous fluid, which becomes turbid as a result of secondary infection. When the bullæ become septic, there is a hectic temperature and pricking sensation due to pus formation.

TREATMENT:—Externally calamine lotion or cremor zinc is applied to dry up the vesicles. Ointments and dusting powders are both contraindicated as they aggravate the eruptions.

A course of 6 to 10 injections of 5 c. c. of 10% soluseptasine, intravenously every third day will prevent secondary infections. Ten injections I.M., of 2 c.c. each of liver extract twice a week will increase the general resistance.

Usually the eruptions subside within a month with this treatment. If the bullæ still persist, ACTH which is a colloidal solution ready for intramuscular injection is given. Pemphigus requires a higher dose to start with, 25 units every six hours continued until no new lesions develop and the old lesions show healing. The dose is then reduced to maintenance levels and later gradually stopped.

Cortisone in the Treatment of Cutaneous Diseases

Costello treated over 60 patients with 20 different skin diseases, with cortisone. At first, a dose of 200 to 300 mg. was used on the first day, decreasing on the second day and further reduced to 100 to 150 mg. daily from the 3rd day onwards. A safe maintenance dose was between 75 and 150 mg. daily in divided doses. Beneficial therapeutic effects of this hormone and also the dosage, were the same whether administrered orally or parenterally. Costello finds that the principal indication for cortisone was as follows: treatment of severe acute dermatitis venerata, and dermatitis medicamentosa, particularly in severe urticarias and angioneurotic œdema following antibiotic therapy. Good results were obtained in: pemphigus vulgaris, psoriasis arthropathics, erythema multiforme, seborrheic dermatitis, common eczemas; some benefit was seen, during the period of administration (but relapse occurred on the withdrawal of the hormone) in systemic and chronic lupus erythematosus, periarteritis nodosa, dermatomyositis mycosis fungoides, chronic urticaria, scleroderma, and epidermolysis bullosa. No benefit was obtained in psoriasis vulgaris, alopecia keloids, or periadenitis mucosa necrotisans recurrens. Contraindications to cortisone therapy are:—general debility, duodenal ulcers, infancy or old age, essential hypertension, cardiovascular or renal diseases, diabetes, gastric ulcers, intercurrent infection and psychotic states. Mild biologic or toxic symptoms attributable to cortisone were seen in 55% of all patients treated and severe side-reactions in 15 per cent. In the majority of instances all mild and many severe toxic side effects were reversible when the drug was withdrawn.—(*New York. State. Med. Jour.*, 51: pp. 2017-2021, 1951).

MANAGEMENT OF PYOGENIC DERMATOSES WITH ANTIBIOTICS

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ANTIBIOTICS have revolutionised the treatment of bacterial infections of the skin though they cannot completely replace the other conventional forms of treatment. Sulzberger and Baer in their excellent review in the Year Book of Dermatology (1950) have proposed the following criteria for the evaluation of topical antibiotic treatment for pyogenic skin infections :—"Efficacy and spectrum of effectiveness, allergenic potential degrees of systemic toxicity, stability in various vehicles and at various temperatures and cost to the patient ". To these may be added the tendency (1) to cutaneous monoliasis and (2) to the development of resistant strains. The antibiotics that have been tried are :—(1) Aureomycin ; (2) Chloromycetin ; (3) Gramicidin ; (4) Neomycin ; (5) Polymyxin B ; (6) Bacitracin; (7) Terramycin and (8) Tyrothricin.

Unfortunately all these are not available in India and so this discussion is confined to the few that are available here.

Two antibiotics *viz.*, aureomycin and terramycin are in extensive use for the systemic treatment of infectious diseases and they have a low degree of systemic toxicity. Tyrothricin has a strong hæmolytic effect when administered systemically. But these three show no systemic toxicity when applied locally.

Report on the comparative results of treatment by local therapy with the antibiotics :—The antibiotics used were : (1) Aureomycin ; (2) Chloromycetin ; (3) Terramycin and (4) Tyrothricin (Tyroderm). The cases treated were impetigo, Bochart's folliculitis, Sycosis vulgaris, Otitis externa and Furunculosis. The forms of the drugs used were : (1) Aureomycin ointment ; (2) Chloromycetin cream ; (3) Terramycin ointment ; and (4) Tyroderm. The application was made for from 3 to 10 days and the best results were obtained with chloromycetin and aureomycin. Terramycin showed equally good results. The results with tyroderm were disappointing.

The following is a survey of twenty cases of impetigo (five cases for each of the 4 drugs). Aureomycin—3 cured ; Chloromycetin—5 cured ; Terramycin—3 cured ; Tyrothricin—none cured. It may be that the superiority of the Chloromycetin cream was due to the stability and availability in oil in water base.

Systemic treatment with antibiotics.—The antibiotics which have been used in the systemic treatment of pyogenic dermatoses are Penicillin, Streptomycin, Chloromycetin, Terramycin and Aureomycin. Penicillin is the antibiotic of choice in acute staphylococcal and streptococcal infections of the skin. This drug is relatively

inexpensive but has a great tendency to form resistant strains. Therefore, the broad spectrum antibiotics, Chloromycetin, and Terramycin are useful in the treatment of penicillin-resistant gram-positive and gram-negative bacillary infections and are conveniently given by mouth. They have a profound effect on the bacterial flora of the stool and interfere with the absorption of vitamins, especially the B-complex and disturb the normal balance between the bacteria and yeast organism.

The side-effects of these drugs are :—Nausea, vomiting, diarrhœa, anorexia, perianal pruritus and stomatitis. The advantage of these antibiotics lies in their broad-spectrum activity. Streptomycin is rarely used in the systemic treatment of cutaneous bacterial infections. Some dermatologists consider that penicillin O is less allergic than penicillin G, but I have no experience of this drug.

Summary.—Topically, chloromycetin cream and systemically, penicillin continue to remain the antibiotics of choice. Cases which are resistant to penicillin may be treated with Aureomycin, Chloromyeetin or Terramycin orally. There is nothing to choose between these three. Vitamin B-complex should be administered concurrently.

It must be realised that antibiotics have revolutionised the treatment of pyogenic dermatoses, but have not replaced the other therapeutic measures such as starch-baths etc. Due care is necessary in the treatment of predisposing factors like anæmia, diabetes, focal sepsis or other concurrent infections. In course of time, the use of the other new antibiotics Bacitracin, Gramicidin, Neomycin and Polymyxin B not now readily available here, may further enlarge the scope of dermatological therapeutics.

185, Vakil New Street, Madurai.

Bacitracin ointment in the treatment of Pyogenic Infections

Wrong and his colleagues treated 27 cases of various types of pyodermas with a bacitracin ointment containing 500 units of bacitracin per gram of the base, *viz.*, wool fat, white mineral oil and white petrolatum. The duration of treatment ranged from 9 to 30 days. Excellent results were obtained in six, good results in 16 others; only five did not respond. The successful cases consisted of those suffering from *Staph-pyogenes* infections such as :—Impetigo, folliculitis of the beard, pyoderma blepharitis and conjunctivitis.—(*Canadian Med. Assoc. Jour.*, 64: May 1952).

COSMETIC DERMATOLOGY

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SOME one said that civilisation means syphilisation. As civilisation advances, there is a large increase in the use of cosmetics. Cosmetic dermatology relates to the dermatitis caused by allergy to cosmetics. At the present day both men and women use cosmetics and the incidence is the same in the two sexes. The products which cause sensitisation include hair dyes, hair-tonics, hair-lotions, lip-sticks, nail-polishes, base coats, freckle creams and shaving lotions and creams.

Dr. Norman Tobias classifies the causes of cosmetic dermatitis under three headings *viz.*

A. Errors and omissions on the part of the patient.—(1) Instructions issued by the manufacturer are not strictly followed. (2) Using cosmetics on an already irritated skin. (3) Too much, too frequent and vigorous use of the cosmetics. (4) The use of cosmetics on areas exposed to sunlight.

B. Defects in the cosmetic.—(1) The use of old and badly stored cosmetics. (2) The cosmetic may be unstable due to the absence of stabilisers or emulsifiers. (3) The cosmetic may contain potential sensitizers. (4) The use of concurrent antibiotics and chemotherapeutics may induce cross-sensitization. (5) Chemical changes might arise in the cosmetics producing secondary irritating substances. (6) The cosmetics may be contaminated by minute amounts of powerful sensitizers.

C. Errors and omissions on the part of the manufacturer.—These could be avoided if helpful co-operation exists between the dermatologists and the cosmetologists. The art and practice of cosmetology do not mean merely spectacular booms in the sale of these products but the producion of cosmetics which do not cause dermatitis. The defects may be as follows :—(1) The production of a cosmetic without proper and controlled medical research and (2) the substitution of cheaper goods.

The commonest causes of dermatitis.—Two cases of lip stick dermatitis have come to my notice. A good lip stick (1) should be capable of being easily and evenly applied ; (2) should not be too greasy ; (3) should not sweat ; (4) should not crumble or crack ; (5) should have an agreeable and pleasant smell, and taste and the colour must be uniform.

Lip sticks consist of wax, fat and an oil base in which are blended oil–soluble dyes and pigment colours. Sensitivity to pigment colours is rare, but sensitivity to dyes is fairly common.

The waxes commonly used are :—(*a*) Beeswax. (*b*) Paraffin wax. (*c*) Ceresin. (*d*) Carnauba. (*e*) Spermaceti. The fats used

are :—(1) Cocoa butter, (2) Adeps benzoate, (3) Cetyl alcohol (4) Wool fat, (5) Hydrogenated vegetable oil, (6) Vaseline, (7) Cholesterol absorption bases. The oil-bases commonly used are :—(1) Mineral oil, (2) Soya-bean oil, (3) Peanut oil, (4) Sesame oil, (5) Castor oil, (6) Butyl stearate.

Natural and synthetic flowers and essential oils are used for taste and colour.

Lip-stick-dermatitis is characterised by swelling and œdema of both lips, scaling, fissuring, erosions and vesiculations. Subjective symptoms usually are burning, tingling and dryness. The symptoms might appear when the patient takes to a new type of lip-stick or the continued use of the same brand of lip-stick for months or years. The disease is to be differentiated from other local conditions as herpes, angioneurotic œdema, chancre, eruptions due to drugs, malignant conditions and ariboflavinosis.

Cosmetic dermatitis is a form of allergic eczematous contact-dermatitis. The eruptions usually disappear soon after discarding the offending agent. In the case of hair dyes producing cosmetic dermatitis, hospitalisation is necessary if permanent loss of hair and secondary infections are to be prevented.

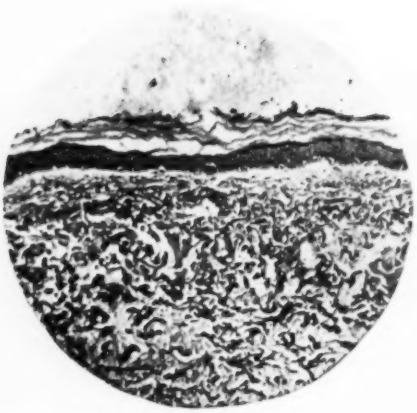
Treatment for cosmetic dermatitis.—The treatment is similar to any other form of allergic eczematous contact-dermatitis, *viz.*, (1) discontinuance of all cosmetics, (2) local treatment for acute or subacute conditions. In the acute stage use wet compresses with boric lotion or alumium acetate lotion. After the acute stage subsides zinc-cream may be used. After sometime the cosmetic preparation must be changed, as it has come to stay as a necessary evil. Some cosmetologists have testing sets for various cosmetics and when available they may be conveniently used for a patch test by the attending physician.

Lichen Planus of the Scalp

No specific treatment is indicated. Internal measures include the use of heavy metals, such as bismuth, mercury or arsenic. The scalp lesions rarely need local applications although in cases of a severe pruritus, the application of a fifty per cent trichloracetic acid solution directly to the lesions will often result in improvement. This application requires neutralization by sodium bi-carbonate solution after a two-minute period. This improvement, of course, is preceded by a short period of irritation at the site of application.—(Behrman in *The Scalp in Health and Disease*, 1952, p. 432).

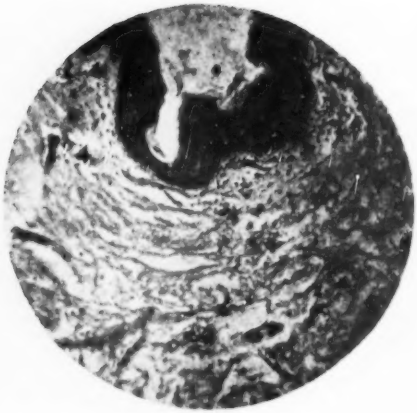
DERMATOMYOSITIS

A. V. S. Sarma and S. Krishnan.

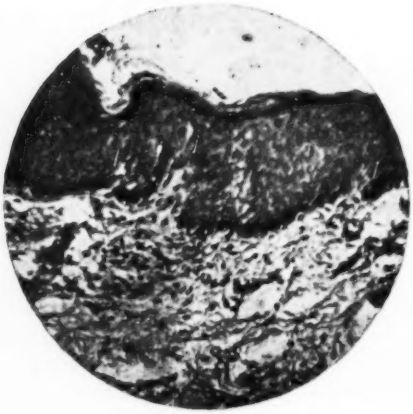


Skin x 160

Skin x 320



Skin-muscle x 160



Skin-muscle x 320

DERMATOMYOSITIS

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DERMATOMYOSITIS is characterised by fever, œdema, multiple myositis and dermatitis and courses through a period of two years.

The onset is rarely acute and usually insidious with febrile debility. The muscles are inflamed and tender, with local or generalised œdema, and dermatitis with erythematous or urticarial eruptions and sweating. The spleen may be palpable. The muscles atrophy with fibrosis and develop contractures. Debility and involvement of respiratory muscles conspire to bring about terminal pulmonary infections.

Dermatomyositis and scleroderma probably represent links in the same morbid process. The corresponding skin is swollen, hard and œdematous while the muscle is yellow and œdematous with lymphocytic infiltration and getting into degenerated areas.

TREATMENT: is symptomatic and glycine is reported to give good results. An illustrative case is elaborated below.

An adult female aged 22 years, Hindu, Brahmin, vegetarian was admitted into the Government Royapettah Hospital on 1-7-'52 for œdema of the lower limbs of one month's duration, and pain in the back and legs for two weeks. She was discharged on 8-10-'52.

Family history:—Has two sisters aged 30 and 28, both healthy; mother aged 54, healthy; father died 10 years ago when he was about 55. The patient is married but has no children. Clinically, the patient is slightly pale and walks with flexion of hips and knees on either side. Heart: nil particular. Lungs: nil particular. Abdomen: soft. Liver: just palpable. Spleen: not palpable. A slight œdema of the face and neck was observed as periodic manifestations while the patient was in the ward for about three months. A "V" shaped area of hyperpigmentation was present on the upper and central part of the back. Muscles atrophied and of poor power could be felt hardened (fibrosed); particularly evident around the left knee joint, this was such as to limit movements.

Nervous system:—Memory and intelligence were good, but was mentally depressed as a result of poor muscular power. There were no signs of myelopathy.

Investigations done:—Urine showed no sugar or albumin. Urine Creatinine 128 mg. per cent present. Blood: Kahn and Wassermann: negative. Basal metabolic rate: negative 8 per cent. X-rays: Pelvis: no bony lesion. Ankle joints: no bony lesion.

Dorsi-lumbar spine: no bony lesion. Chest: lung fields clear. (Appearance of slight rarefaction of bones present).

Biopsy of skin and skin-muscle clip :—By Prof. D. G. Reddi, M.D., Professor of Pathology, Madras Medical College, Madras.

Microphotographs of the skin tissue :—The epidermis is flattened with insignificant papillæ. The connective tissue fibres of the corium are transformed into enormously enlarged coarse fibres and run parallel to the surface epithelium in wavy bundles. There is marked reduction in the sebaceous and sweat glands. Hair follicles are absent and a few surviving blood vessels are sclerosed. (*Vide two photographs* x 160 and x 320).

Microphotographs of skin-muscle clip: —The photographs show only epidermis and subepidermal connective tissues. Changes in the muscle fibres are not visualised. There is atrophy of the epidermis in areas. The corium is œdematous, particularly in the upper layer. The connective tissue fibres are swollen and opaque. Under high power magnification, fragmentation of some of the fibres is seen. There are a few round cell collections. There is absence of sebaceous and sweat glands. In fact the changes have some resemblance to those of scleroderma. A few muscle fibres that are seen are atrophic, fibrotic connective tissue replacing the muscle bundles. Evidently this is an old lesion where fragmentation of muscle fibres and nuclear changes will not be present (*vide two photographs* x 160 and x 320). (It was with great difficulty that the biopsy of muscle could be got and the scalpel was taken by the surgeon as deep as the iliac bone with the idea of getting a clip of gluteus maximus, and what was got was plenty of fat mass with a tug of a few muscular fibres only).

TREATMENT :—Orally multivitamin pellets and a recipe containing Sodii salicylate were given, also vitamin B complex by the intramuscular route. Vitamin E and thyroid extract were tried orally for one month. Treatment, if effective, was probably due to suggestion.

Conclusion.—The above study is on Dermatomyositis, a condition which is receiving greater attention, at the present day.

Our thanks are due to Dr. M.A. Latif Khan, our Superintendent for his uniform encouragement. We are thankful to Lt.-Col. C. K Prasada Rao, M.D , Dean, General Hospital, and specially to Dr. D G. Reddi, M.D., for his valuable reports, and supply of microphotographs on the biopsies; and also to Dr. T. V. Jacob, Honorary Assistant Surgeon of our hospital for obtaining the biopsy material for examination.

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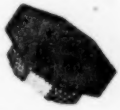

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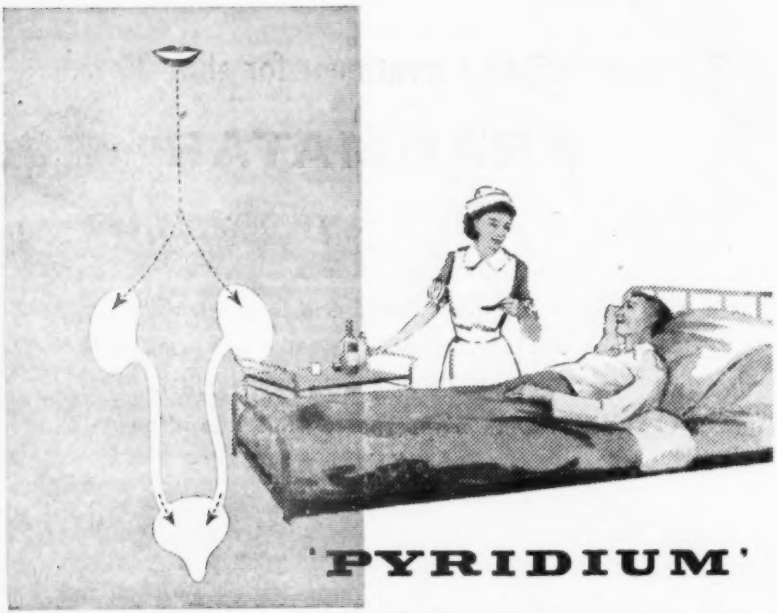

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CLASSIFICATION OF LEPROSY*

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EMINENT leprologists have been trying to make the classification of leprosy as scientific as possible and they have put forward clinical, bacteriological, immunological and histological reasons for different groupings, but unfortunately these cannot be correlated in all cases of the same group. Secondly, while making finer differentiation of clinically similar lesions, they put forward histological and immunological reasons which the ordinary workers cannot apply as they have to depend upon bacteriological and clinical findings only. Thirdly, there are some objections with regard to terminology.

At the Manila Conference, leprosy was divided into two types —'Neural' and Cutaneous'. The latter term was objected to and at the Cairo Conference it was replaced by the term 'Lepromatous'. Objections still remained because the term 'Neural' was topographical and the term 'Lepromatous' was histological. The erythematous thick patch which was not included in the Manila classification was included in the Cairo Conference under the neural type and it was called 'tuberculoid'. Subsequently this was considered a polar type in the South American classification, in which leprosy cases were grouped under three headings, lepromatous, tuberculoid and uncharacteristic. It is stated that the tuberculoid type does not pass on to the lepromatous type. When such a transformation was reported by some eminent leprologists, others equally eminent refuted that they were not typical tuberculoid cases, the histology being slightly atypical or the lepromin test being weakly positive. But according to yet other eminent leprologists "the 'borderline' group arises from tuberculoid leprosy and occasionally may develop until it is quite indistinguishable from cases which were lepromatous from the onset." The term 'tuberculoid' is objected to by some workers and the term 'lepride' is suggested instead. Others are of opinion that the suffix 'ide' is used in dermatology to indicate allergic lesions like tuberculide and that an erythematous, thickened patch of leprosy is not an allergic skin lesion. Hypopigmented flat patches which were called 'simple' lesions in the Cairo classification were changed to 'uncharacterisitic' in the South American classification, which was then changed to 'indeterminate'. According to some workers this term 'indeterminate' is open to serious objections and they suggested division of this type into two groups—lepromin negative cases to be called 'pre-lepromatous' and lepromin positive cases as 'tuberculoid'. Some workers' consider that there is no such lesion as 'indeterminate' from the histopathological point of view, as even the earliest lesions are either potentially lepride or a

* This article is based on a paper read at the 4th All-India Leprosy Workers' Conference held at Puri in January '53.

potential leproma. But unfortunately most leprosy workers have no facilities to do the lepromin test or make a histological study.

In the All India Conference held at Wardha (1947) Dharmendra remarked very consistently that histological findings are not a constant feature in all cases of the same type, and in all stages of activity of an individual case and that facilities for histological work are not available to a vast majority of leprosy workers who have to depend for diagnostic classification on clinical considerations. At the Calcutta Conference (1948) the same view was reiterated by Dharmendra that a particular histological picture, with perhaps the exception of the lepromatous histology was not confined to one particular clinical variety of the disease, and that it was therefore, wrong to use histopathological terms as synonymous to indicate the various clinical types of the disease. In his opinion the use of histological terms to indicate clinical types resulted in confusion in many instances. Difference of opinion also exists as regards results of the lepromin test. Some consider that the lepromin test should be positive in all tuberculoid cases. But in actual experience it was found to be positive only in 70% of the tuberculoid cases. The negative results were considered to be due to faulty technique. Lately there has been a suggestion that the negative or weakly positive cases of the group are possibly 'borderline' cases. Some consider that a definite positive reaction never becomes negative, others fail to corroborate it in many cases. The lepromin test, usually negative in almost all lepromatous cases, was still found positive in some.

As regards thickening of the nerves, some believe that thickened nerves are not seen in lepromatous cases, though it has been commonly found in Calcutta. Again difficulties exist with regard to the classification of cases that are neither typically tuberculoid nor typically lepromatous. Recently there has been a suggestion that these cases may be called 'dimorphous' and may be divided into two types, (*a*) those which still show a predominantly tuberculoid feature (DT), and (*b*) those which show a predominantly lepromatous feature (DL). Now the question arises of how to use this histological knowledge in clinical classification. Yet another difficulty in classification is that if the different types are made water-tight by specifying their clinical, bacteriological, immunological and histological characters, a large number of cases of the same type will have to come out as not conforming to the specifications. On the other hand if the specifications are relaxed, it will be difficult to draw a line between them and different workers may classify the same case differently. Leprologists working in different places have different experience and have their own conceptions; so generalisations may not find favour with workers in other places. This will also create confusion in the minds of the ordinary workers. Again it is not always possible to legislate for all variations. In order to

overcome these difficulties and make matters simple and practical, I proposed a new classification at the All-India Leprosy Workers' Conference, held at Warda in 1947. This was entirely clinical (inculding bacteriological) and had for its main object, uniformity and ease of application by all leprosy workers. But as it did not accord with the International classification, it did not receive universal general approval. The present trend is that a classification to be acceptable should conform in general to what was proposed at the Havana Congress. An approach to this problem is attempted from the practical side by citing cases of the different types. Typical and atypical cases and the clinical characters and bacteriological findings are set out in detail for the benefit of the general workers.

Lepromatous type (L).—Sub-classification L1, L2 and L3. This term has been generally accepted. In lepromatous cases the clinical and bacteriological findings may be :—

Lepromatous cases of group (*A*) :—1. Generalized nodulations and infiltrations, no anæsthesia, no thickened nerve, no deformity. Smears strongly positive. 2. The same as (1) but in some parts there may be patches which may be hypopigmented, and flat or red and thick, smooth and non-anæsthetic. Smears same as in (1). 3. There are no nodules but only generalised diffused infiltrations all over the face, ears, body and extremities. No anæsthesia. No thickened nerve. No deformity. Smears same as in (1). 4. Same as (3) but in some parts there are patches which may be hypopigmented and flat or red, thick, smooth and non-anæsthetic. Smears same in (3). The question then arises as to what should be the classification if :—(*a*) the lepromin test be positive in some of the above cases ; (*b*) the histology be not typical in some respects ; (*c*) anæsthesia is present in some of the patches of cases under (2) and (4) ; (*d*) multiple thickened nerves be found and if the thickening be considerable or if there be a nerve abscess.

Examples :—(1) S. K , a patient of Albert Victor Hospital, Gobra, Calcutta was a L_2 case. He developed multiple subcutaneous swellings which were suspected to be suppurating subcutaneous nodules or pseudo-neurofibromas. Ultimately one swelling on the radial border of the right wrist was exposed by operation and found to be a nerve abscess in connection with the superficial radial nerve which was thickened. On further examination other swellings were also found in connection with nerves which were slightly thickened. (2) Case No. 7098. Classification L_3. Chart 3. Big nodules and diffuse infiltrations in ears, face, body and extremities. Lt. ear +++, Lt. arm ++++, Nose ++. Considerable thickening of both ulnar nerves, and their dorsal branches, superficial radial, lateral popliteals and superficial peroneals.

(*e*) if extensive anæsthesia is found in extremities besides generalised nodulation and infiltrations.

Example:—Case No. 6554—Lz. Chart 8. Stocking-like anæsthesia in legs and feet. Smears—Lt. ear +++. Lepromin—nil. Section—leproma.

(*f*) if extensive, anæsthesia in extremities, deformities, paralysis, trophic ulcers are found, besides generalised infiltrations and nodulations in some parts.

Example:—Case No. 9125. Chart No. 1. Extensive anæsthesia in hands, forearms, arms, feet, legs, thighs. Deformities of the finger of both hands. Smears—Rt. ear ++++, Lepromin—nil. Section—leproma.

Comments:—In my opinion even if there are some atypical immunological or histological findings these cases should be called lepromatous. Neuro-anæsthetic signs found in a lepromatous case do not warrant a changing of the main classification of 'lepromatous' but the neuro-anæsthetic signs should be indicated by proper symbols. A lepromatous case with no neuro-anæsthetic signs but only thickened nerves need not be indicated by any special symbol. Thickening of nerves or nerve abscesses do not always indicate a tuberculoid change.

Lepromatous cases of group (*B*):—There are some cases which may not have diffuse infiltrations or nodulation but have only one or more localised patches which may be hypopigmented and flat, or red, thick, smooth and sometimes ill-defined. There is no sign of lepra reaction, and anæsthesia or thickened nerves may or may not be present. But contrary to expectations, smears are moderately (++) or strongly (+++) positive. The real nature of the lesions of some of these cases may be clarified by the immunological test and by histological examinations, while in others the doubt will not be clarified.

Comments:—Outwardly they look like bacteriologically positive neural macules of the Cairo classification. But histologically some of them are found to be lepromatous with the lepromin test negative while the findings are different in others.

Lepromatous cases of group (*C*):—When the cases under Group (B) improve under treatment, red and thick patches subside and become less thick and red. Fewer bacilli are found in the smears and ultimately only very few bacilli may be found (+)*. But the patches which were originally lepromatous still remain so, but the lepromin test may be negative. Others have different histological pictures and immunological results. When a case of this type is seen for the first time and when the patient cannot give an

* + Bacilli not in every field, but one or two in some fields, out of the 50 fields examined.
++ Small number of bacilli in almost every field and some globi in 50 fields.
+++ Many bacilli and some globi in every field.
++++ Every field is full of bacilli and many globi.

accurate history, it is sometimes difficult to decide whether the patches seen are partly subsided lepromas or borderline lesions or minor tuberculoid lesions, smears from which are still slightly positive. The results of lepromin test are not always helpful in these cases. Similar difficulties may arise in cases where the lesions are hypopigmented and flat but the smears are slightly positive. Such lesions may be neural, simple, bacteriologically positive lesions or completely subsided lepromatous, or 'borderline' or reacting tuberculoid lesions. We have to decide about the classification in these cases.

Tuberculoid (T) :—Sub-classification T_1 T_2 and T_3.

The term tuberculoid has come to stay. In typical cases these lesions are quite red and raised so that they have well defined margins. The surface is rough, dry, and sometimes scaly. If the lesions be on the face and trunk, they may not be definitely anæsthetic but analgesia will be present. Nerves supplying these patches are frequently found thickened. In the reactional phase, some patches may ulcerate or become œdematous. Smears are usually negative or slightly positive. Lepromin test is usually strongly positive and the histology is of the tuberculoid type. There is no difficulty in classifying these cases. Due to involvement of the nerve trunks, neuro-anæsthetic lesions may be found in some cases which should be indicated by proper symbols.

Comments :—Sometimes the histological and immunological findings may not be quite typical and still these cases should be considered tuberculoid. As already stated in some cases, the lesions may resemble tuberculoid patches but the smears may be moderately or strongly positive, and the results of the lepromin test may be variable, *i.e.* positive in some, but doubtful or negative in others. Histology may be tuberculoid with some atypical findings, double histology of both tuberculoid and lepromatous, no definite tuberculoid or lepromatous change or the histology may be definitely lepromatous (Table 1). Clinicians always find difficulty in classifying these cases, because it is difficult to ascertain from clinical findings what the histological picture may be like. Lepromin results do not always tally with the histological findings. For example, the lepromin test may be positive but the histology may be lepromatous (See Table 1. Case No. 9402). In the further advanced condition these lesions become succulent and smooth and the margins become ill-defined. Lepromin test may be negative. At this stage, it can be classified as lepromatous with more certainty. All these cases with bacteriologically strongly positive muscular lesions, whether the histology is typically lepromatous or not and whether the lepromin test negative or not at the beginning, soon become generalized lepromatous cases clinically (like cases under Group A) as well as in other respects.

TABLE I

Erythematous, thick and smooth localised patches

Case No	Clinical classification	Smear result	Lepromin test	Histological findings	Subsequent changes
8019	Neuromacular positive.	+ + +	Doubtful	Doubtful	Later the patient became clinically lepromatous having generalised infiltrations and erythematous smooth macules. Section—Leproma.
8133	Do.	+ +	Positive	Do.	Later the lesions subsided under treatment.
7896	Do.	+ +	Do.	Tuberculoid.	Discontinued treatment.
9402	L^2Na^2. Lepromatous case with neuro anæsthetic signs.	+ + +	Do.	Leproma	Lesions subsided under treatment. Smear from only one place slightly positive.
8903	Doubtful (N?L)	+ +	Negative	Do.	Later clinically lepromatous.
8821	Neuromacular positive	+ +	Do.	Do.	Lesions subsided under treatment. Smears slightly positive in two places.
9767	Do.	+ +	Do.	Doubtful ? Tuberculoid reaction.	Improving under treatment.
8040	Doubtful (N?L)	+ + +	Positive	?Leproma.	Later clinically lepromatous. Section—Leproma.
8831	Do.	+ +	Do.	?Tuberculoid reaction.	Lesions subsided under sulphone treatment. Smears negative.
6736	L2 (Numerous small lesions).	+ + +	Negative	Both tuberculoid and lepromatous histology.	Later typical lepromatous clinically. Section—Leproma.

Example:—Case No. 7625. Clinical classification : Neural—positive case having localised red and thick patches.

Chart No. 1. Fig. 1 (*Vide page 505*).

3-4-'44 Smear + +,	Lepromin	Erythema 19 Induration 5	+ ; Section—not done
24-4-'44 —	Lepromin	Erythema 23 Induration 5	„ ; Section—not done
30-7-'44 Smear—Neg.,	Lepromin	Erythema 26 Induration 6	„ ; Section—not done
18-3-'44 Smear—Neg.,	Lepromin	Erythema 30 Induration 3½	„ ; Section—not done

Chart No. 6. Fig. No. 2. (*Vide page 506).* 7-5-'51. Returned after 4 years. Nodules and infiltrations. Classification—L_2. Smear + +, Lepromin—nil. Section—leproma.

It is to be noted that smears which were positive at the beginning, became negative within 16 months and lepromin test was positive four times within the first two years. But still the case became lepromatous later. Two other similar cases (No. 7368 and 7098) were also seen.

Therefore, these cases are either lepromatous or allied to it and should be classified as lepromatous notwithstanding any atypical immunological or histological findings at the time of the first examination. This will eliminate the need for a 'borderline' group and remove most difficulties experienced by clinicians. In these cases the bacteriological findings are the most important for classification.

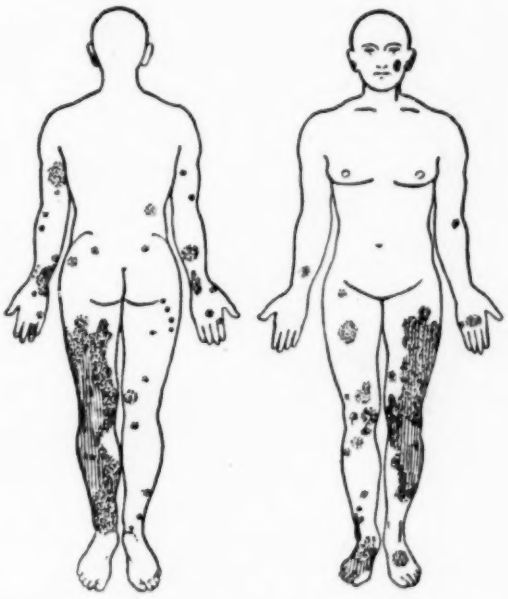

Fig. 1. Case No. 7625 Chart No. 1
Localised red and thick, anæsthetic patches.
Nerves thick. Smears + + Lepomin—$\frac{\text{Erythema } 19}{\text{Induration } 5}$ +
Section—Not done [*Vide page 504*

There are other cases where, due to repeated reactions, the positivity of smears increases. Patches may resemble lepromatous ones and anomalous histological and immunological findings may indicate that the cases on the whole are approaching the lepromatous type; or there may be actual conversion into the lepromatous type. Therefore, when the smears are strongly positive and clinically the patches appear to be more like lepromatous being smooth and shiny, they are best considered lepromatous.

Simple, uncharacteristic, indeterminate.—None of these terms have received final approval. These lesions may be called undifferentiated (U) in contrast to the differentiated lesions (Tuberculoid and Lepromatous). Subclassification may be U_1, U_2 and U_3. These lesions are hypopigmented and flat but not erythematous

(when erythema is found the histology is usually minor tuberculoid). The loss of pigment may be variable. In some cases these lesions may be very faint and ill-defined, and anæsthetic when found in the extremities. Lesions on the face and trunk are not always anæsthetic, but usually analgesic. Smears are negative or slightly positive(+) in a few cases. Thickened nerves may be found in association with these lesions and consequently there may be acroteric lesions in the extremities. Lepromin test is usually positive but may be negative or doubtful in some cases. Histology is either simple or minor tuberculoid. Up to this stage, there is no difficulty in classifying these cases as undifferentiated.

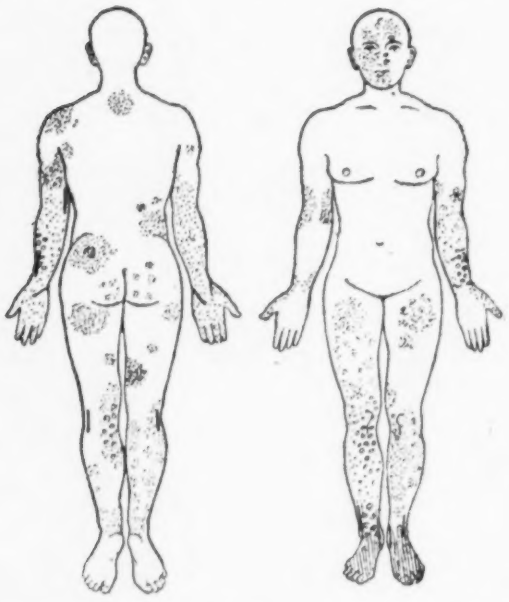

Fig. 2. Case No. 7625 Chart No. 6
After seven years. Nodules infiltrations in different parts.
Nerves thick. Smears + + Lepromin—Nil
Section—Leproma [*Vide page 505*

COMMENTS:—In some cases though there may be some atypical immunological or histological findings they should be classed as undifferentiated so long as the lesions remain flat and the smears negative and there is no clear indication clinically of a tuberculoid or lepromatous change.

A lesion may be hypopigmented from the very beginning or after subsidence of a tuberculoid or lepromatous patch. Sometimes it is difficult to determine from the history what the previous nature of the lesion was. Therefore, for the sake of simplicity all bacteriologically negative cases having hypopigmented, flat patches should be considered undifferentiated. It will not always be possible to classify these lesions as resolved tuberculoid lesions, resolved leproma, or early maculo-anæsthetic lesions, unless the patient is under close observation for a long time.

Sometimes there may be one or more hypopigmented patches the smears from which may be moderately or strongly positive. It then becomes difficult to ascertain whether it comes under this

group, progressing towards the lepromatous type and now a 'borderline' case, or the patches are really lepromatous or 'borderline' patches which were erythematous and thick before.

Example:—Case No. 6554. Clinical classification was doubtful (N?L)

Chart 1. Hypopigmented multiple patches. Fig. 3. No nodule or infiltration. Smear + + + Lepromin—nil. Section—early leproma

When hypopigmented patches improve further, they may still retain their clinical character or become ill-defined. There is reduction in the number of bacilli and smears may be slightly positive. (+). It is again difficult to determine whether it is an undifferentiated positive case, or a subsided lepromatous or borderline case, or a reacting tuberculoid case in whom the patches have now subsided and smears are only slightly positive. The histology of these lesions may be simple, minor tuberculoid, lepromatous or doubtful, Lepromin test is usually negative. (*Vide picture above*).

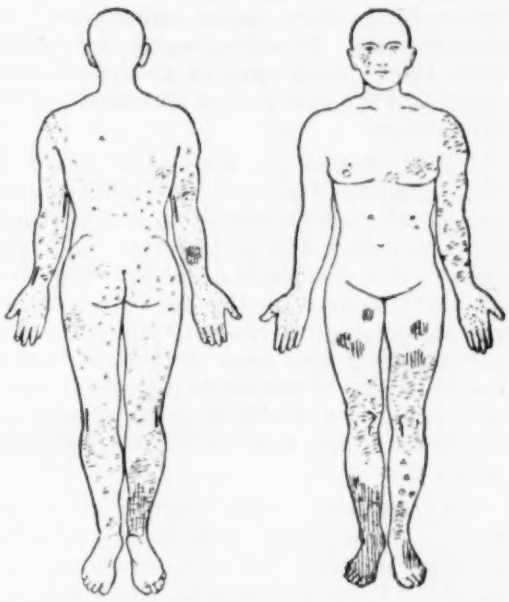

Fig. 3. Case No. 6554 Chart No. 1
Only hypopigmented, multiple flat patches.
Some of them anæsthetic. (No nodules or infiltrations)
Nerves thick. Smears + + Lepromin—Nil
Section—Leproma.

When lepromatous patches subside further, skin smears may become negative while the nasal smears still remain positive. What should be the classification if the patient is seen for the first time at this stage? We saw some neural cases (of the Cairo classification) where smears from skin lesions were negative but smears from the nasal mucous membrane were positive. There should be some means by which these cases can be classified.

Neuro-anæsthetic or neural type (N).—Subclassification N_1, N_2, and N_3. In this type some or all of the following signs may be present: (1) Anæsthesia—There may be circumscribed areas of anæsthesia in the distribution of a thickened nerve without any macular lesions, or there may be glove or stocking-like anæsthesia

and thickening of the nerve supplying the anæsthetic parts. If there be only anæsthesia with no thickening of the nerve and no other signs of leprosy, the diagnosis and classification should be postponed and differentiation should be made from other conditions which may cause anæsthesia, *e.g.* Bernhardt's syndrome, diabetes, etc. If subsequently a hypopigmented or an erythematous thick patch appears at the same site, diagnosis can then be made and the case can be classified according to the nature of the skin lesions.

A thickened nerve may be found in all the principal types (Lepromatous, Tuberculoid, Undifferentiated). The Cuba conference suggested their classification into polyneuritic leproma, polyneuritic tuberculoid and polyneuritic indeterminate. It is difficult to classify the thickened nerve into the three main types depending on the histology of the nerve which is usually impossible, and on the immunological reaction which is not always consistent. It may be confusing also because the skin lesions may be of one type and the histology of the nerve may be of another type and the lepromin results may be doubtful. (2) Anæsthesia and wasting of muscles. (3) Anæsthesia, wasting of muscles, deformities, paralysis, trophic ulcers etc.

COMMENTS:—Some leprologists suggested the word 'polyneuritic' for these cases. The term polyneuritic means involvement of multiple nerves. But in some cases of leprosy only one nerve may be found thickened causing anæsthesia, deformity, paralysis, trophic ulcers, etc. in one limb. Therefore, these lesions are better indicated by the letter 'N' to denote neuro-anæsthetic manifestations.

One leprologist suggested a further classification of these cases into primary and secondary. According to him, primary cases are those "which are without skin lesions or residual evidence or histories of such as cannot consequently be assigned to any of the primary classes, and yet present changes referable to involvement of nerve trunks." "Secondary cases would be those which in the preceding active phase, were of any of the primary classes with polyneuritic changes which persisted as permanent residue after the active disease had died out." Sometimes it is difficult to determine from the history what the previous nature of the lesion was. It may not be possible always to determine whether the hypopigmented flat patches at the time of examination of a patient in a clinic, are residual macules or active macules of the undifferentiated type and whether a case having polyneuritic deformities is an old inactive case or an active case with extensive hypopigmented patches, thickened nerves, deformities, etc. Secondly, there may be a tuberculoid lesion on the forearm causing thickening of the ulnar nerve and later still, there may be anæsthesia and deformity in the hand. Evidently the neuro-anæsthetic lesions in this case are secondary to the skin lesion. But according to the definition

given, it is *not* secondary because the active skin lesion is still there. It is not primary because it is not purely neural. Therefore, to avoid confusion and to minimize the number of symbols, cases having neuro-anæsthetic signs may be indicated by the letter 'N' whether these signs are primary or secondary. When these are found in any of the cases under the main classification, the letter 'N' may be placed after the symbol of the main classification. Depending upon the extent of anæsthesia and damage done to a particular part, the neuro-anæsthetic cases may be sub-classified into N_1, N_2, N_3. Very rarely there may be only some thickening of a nerve without any anæsthesia, deformity or macular lesions. If the thickening be definite and proved to be due to leprosy (*i.e.* if bacilli are seen in smears from nerve-fibres) the case may be placed under this group 'N'. Otherwise the patient should be kept under observation.

Polar types.—I shall now discuss the term 'Polar' used in the South American classification and the methods of symbolisation. According to the South American classification, by polar types are meant tuberculoid and lepromatous types. They are called 'polar' because clinically, bacteriologically, immunologically and histologically they are distinct and separate from one another. This is true in typical cases but in many clinically tuberculoid and lepromatous cases, there may be some atypical immunological and histological findings. Secondly, the two poles should never meet. Here we find that from simple or undifferentiated lesions two forms are differentiated—Tuberculoid and Lepromatous and after their subsidence they become undifferentiated again. Thirdly, in the 'borderline' cases the double histology of tuberculoid and lepromatous may be found in the same

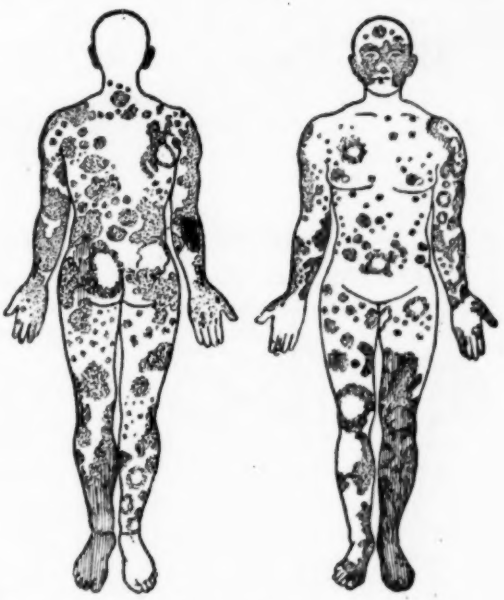

Fig. 4 Case No. 7368 Chart No. 1

Erythematous and thick, multiple patches. Some of them anæsthetic. Slight thickening of nerves. History of reactions in every winter.

Lepromin (Erythema 14 / Induration 4) + Smears (Lt. forearm 1/50 / Rt. arm –Negative

Section—Not done [*Vide page 510.*

patient or in the same section. Therefore, they meet at three points.

It is too early to state definitely that no tuberculoid case can become lepromatous. Our impression is that it is not a common finding. In a typical case where the smears are negative, the lepromin test is strongly positive and the histology is typically tuberculoid, such a change may not take place. But when the smears are positive, the conversion to lepromatous type may take place in some cases after a long time. The larger the number of bacilli in the smears, the quicker may be the conversion. We have seen cases with tuberculoid lesions at the beginning with slightly positive smears and positive lepromin test turning lepromatous after some time.

Example:—Case No. 7368. Neural case having erythematous and thick patches, with slight signs of reaction. History of reaction every winter for the last three years.

Chart 1 (Fig. 4, *vide page 509*). Smear from left forearm 1/50. Smear from right arm—negative. Lepromin — Erythema 14, Induration 4 positive. After one year —Lesions subsided considerably, smears slightly positive. Lepromin–Erythema 20, Induration 4 positive.

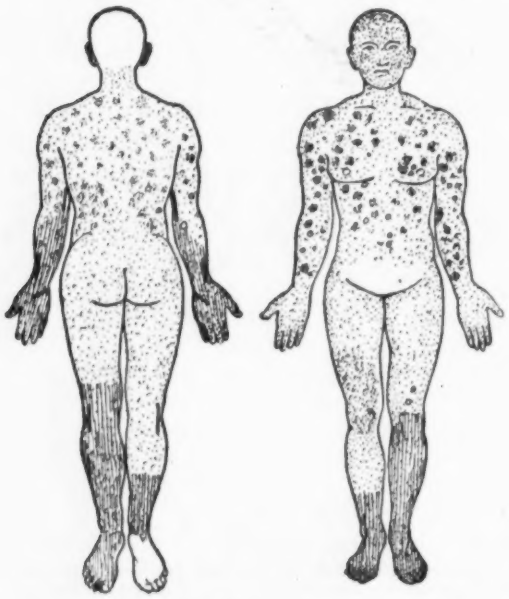

Fig. 5 Case No. 7368 Chart No. 5

7 years after he left treatment. Nodules and infiltrations appeared all over. Nerves were more thick than before. There was anæsthesia in extremities.

Smears (Rt. ear + + +, Nose + +) Lepromin—Nil. Section—Leproma

He was without treatment for 7 years, and then returned. Chart 5. (Fig. 5) shows the present condition:– Lepromatous case. Nodules and diffuse infiltrations all over the face, ears, body and extremities. Smears Right ear + + +, Right nose + +, Section—leproma, Lepromin—Slight erythema, Slight induration – Negative.

It is better therefore, to consider them as two types or two forms and drop the word 'polar. It is too early to assert that a tuberculoid case cannot became lepromatous.

The principle of symbolisation.—The symbols of the three main forms have already been indicated and it has also been suggested that neuro-anæsthetic signs found in any of the three main types should be indicated by the letter 'N'. Where there are no visible skin lesions of any of the main types but only some neuro-anæsthetic signs and thickened nerves, the letter '*N*' *alone* should be used to denote the case. According to the number and extent of lesions and damage done to the affected parts, the cases may be sub-classified by adding 1, 2, or 3 after the main classification like L_2 N_2, etc.

If definite lepromatous lesions are found even in one place the case should be called lepromatous (L) although the lesions in other places may have subsided and become undifferentiated or when other lesions are not typically lepromatous but doubtful or on the borderline. Similarly, if there be a tuberculoid skin lesion anywhere, the case should be called tuberculoid 'T' although other lesions may have subsided and look flat, as frequently seen in patients having multiple erythematous thick patches. Lesions of the extremities usually subside completely before the lesions of the face, ears and trunk. Bacteriologically positive non-lepromatous cases may be indicated by putting a plus (+) sign after the respective symbols for classification, like T_2 +.

Treatment of Leprosy with Thiosemicarbazone

Barnes treated 10 cases of leprosy with 100 mg. of thiosemicarbazone daily for six months. Of the 5 cases which were lepromatous, 3 improved clinically and the nodules subsided, and lost their shining appearance. No improvement occurred in the other 2 cases. In all the 5 other cases which were tuberculoid, there was well marked clinical improvement. Plaques subsided and lost their erythema, and only faint macules persisted. Improvement was more marked in the tuberculoid than in the lepromatous group ; thus a total of eight of the ten patients therefore, showed clinical improvement within six months of starting this therapy. A further ten cases have since been treated with 50 mg. instead of 100 mg. per day with good results. Barnes considers that thiosemicarbazone is a comparatively milder and less toxic drug but quite effective in treating leprosy, as judged by the results recorded at the leper settlement in Nigeria.—(Barnes, J., *Lancet*, 11-8-1951).

Japanese Leprosaria

The total number of cases in the 13 Leprosaria (12 Government and one private) in December 1951 was 9475 : Lepromatous 63·6%, neural 28·0% ; macular 8·4%. 81 physicians and 319 nurses were employed in these 14 leprosaria *i.e.*, 7 full time doctors per 1000 patients. The capacity of Kikuchi-feifuen, the largest hospital now in Japan is 2160 patients.—(*L. W. M.*, Volume 3).

AN OUTLINE OF THE TREATMENT OF LEPROSY

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THIS article is not meant to be a treatise on the treatment of leprosy, but is written as a practical guide to practitioners, who undertake the treatment of the disease in their private clinics. There are a large number of patients suffering from leprosy who cannot be admitted into a hospital for want of accommodation and there are many others who do not wish to get admitted for various reasons. It may not be possible to treat all of these in a private clinic, but I believe, a fair proportion *can* be treated by practitioners and successfully too.

The first thing to do before undertaking the treatment of a case of leprosy is to establish the diagnosis beyond doubt and it is always advisable to get the diagnosis confirmed by the specialist in the area. Bacteriological examination is also necessary, but here lies the great difficulty. There are no laboratory facilities in the mofussil and one has only to send these patients to a private laboratory in the headquarters town of the District, which involves much expense and inconvenience. Want of these facilities is therefore, a great handicap to the practitioner in the mofussil. I would therefore, enter a strong plea to the Local Boards and Government to make facilities for laboratory examination available in all taluk headquarters hospitals. Having established the diagnosis, the next step is to chalk out the line of treatment suitable for the case and this will depend upon the classification of the case. For, the prognosis, the length of treatment and its success will largely depend upon this classification, and the stage of the disease at which the treatment is begun. I may say at the very outset, that advanced lepromatous cases and neural cases with deformities or chronic ulcers and trophic ulcers with complications must be sent for treatment to hospitals.

Before beginning specific treatment for this disease, it is necessary to have a thorough examination of the general condition of the patient. The teaching of Dr. Muir that the general condition of the patient must be well maintained is a very sound one and I personally attach great importance to it. Anæmia due to worms, specially the hookworm is nearly always present and must be attended to first. The hæmoglobin index of the blood must be brought to a level of at least 60%. The second factor to be looked into is the nutrition of the patient. Advice on this point is essential. Even if he has not adequate means, it is possible to make adjustments in his diet, so that he may get a proper supply of the essential food-factors, especially vitamin B. The third, is to provide some exercise to the patient. A free outdoor life, wherever possible, is to be encouraged or some kind of suitable exercise however mild

should be prescribed. The patient must be made to realise that he is capable of work and this feeling will raise his morale. This occupation will create an intelligent interest in his surroundings and stimulate his recovery. The fourth point I would like to mention is the necessity to assure the patient that success will greatly depend upon his willingness to take the treatment. It is good to tell him at the very start, that the treatment will take a fairly long time and therefore, he must also help in it so as to make it successful. It is good to obtain a definite promise from the patient that he will be regular, follow instructions faithfully and will persevere with the treatment. Sometimes, the fear that the treatment will be long and costly, will deter him from taking it. It is for the practitioner to assure the patient that he need have no anxiety or fear on this account. The next step is to keep a record of the case and charts for this purpose are available or can be improvised. The following particulars must be noted :—name, age, sex, occupation, history of contact and names of any other contacts in the house, duration of disease and classification. The lesions on the body must be marked, so that this chart acts as a guide for future reference and for the progress of the case. Other particulars required are the bacteriological and hæmoglobin index, the weight and other special conditions regarding the case, such as reactions etc. The hæmoglobin index must be recorded every month and the bacteriological index and weight every three months.

Before outlining the details of treatment of leprosy, it is necessary to mention that leprosy is a chronic disease and during its course a number of complications may arise and therefore, the treatment must envisage its complications too. Hence I shall divide the outlines of treatment into two headings : (1) Treatment of the disease ; and (2) Treatment of its complications, which are generally known as reactions.

The treatment of the disease itself first :—The main line of treatment now is the giving of sulphone drugs. The drugs chiefly used are : (*a*) sulphone derivatives ; (*b*) the parent sulphone. Of the sulphone derivatives, Diasone of Abbott & Co., in $\frac{1}{2}$ gramme tablets is given daily in doses of one tablet per day for six days in a week, with rest on the seventh day. The dose is increased from one to two tablets after one month, and may be increased to three tablets in the third month. Sulphetrone of Burrows Wellcome & Co., and Novotrone of Bengal Chemicals, (0·5 g. each) is started with one tablet and slowly increased after two weeks to four to six tablets per day. Diamidine of Parke Davis & Co., is given, similar to Diasone. Injections of Promine of Parke Davis & Co., may be given daily intravenously but the difficulty of giving intravenous injections and the cost involved have to be considered. Injections of sulphetrone prepared by dissolving sulphetrone granules to make a solution of 50% in distilled water and neutralized by the addition of sodium

carbonate in the proportion of 1 c.c. of 10% solution to 5 c.c. of sulphetrone, are equally, if not more efficacious. Injections of sulphetrone are given intramuscularly, especially after reactions. The solution must be fresh and will not keep longer than two weeks.

Sulphone drugs:—The following drugs are available:—Avlosulphone (Imperial Chemical Co.) Cryosulphone (Biddle Sawyer), and Novosulphone (Bengal Chemicals) in tablets of 100 mg. each. To begin with, the drug is given one tablet every other day and after a week or two, one every day with rest on the seventh day. It may also be given only on two days of the week. The total dose should not exceed 800 mg—8 tablets per week. The following precautions have to be taken when giving this treatment. (1) Rest on the seventh day. (2) Hæmoglobin index should never be allowed to go below 60%. (3) The patient must feel better during the treatment, and must not complain of signs of weakness or fatigue. (4) There should be no reactions.

A word must be said about thiosemicarbozone which may be given in doses of 25 mg. increased gradually in the same manner as sulphetrone and which has also given good results. After a course of three months with sulphones, it is necessary to give rest for a period of one month. The treatment with hydrocreol injections intramuscularly or the mixture of esters and olive oil equal parts for intradermal injections is suitable in many cases of neural leprosy, particularly the intradermal injection of a mixture of esters and olive oil. These injections are given on the lesions and give much benefit. A Luerlok syringe fitted with a fine needle may be used for these intradermal injections and is very convenient. The dose is from 1 to 5 cc. and is distributed over the patches and given weekly, but no patch must be injected more than once in two weeks. Hydocreol is given subcutaneously or intramuscularly in doses of 1 to 10 c.c. biweekly. The progress, though slow, is good.

Lepra reactions are characterised by prolonged fever, pain in the body particularly in the long bones, emaciation and prostration. These cases must be distinguished from the minor reactions and the toxic effects which are sometimes produced in some individuals after taking sulphones. The minor reactions consist of fever, pain in the body and a thickening and erythema of existing lesions and the appearance of raised red spots. The toxic effects are weakness, anæmia, which may be severe, dermatitis, neuritis, which is very troublesome, jaundice, and eye affections; mental symptoms are also described, but I have not come across any so far.

The treatment for mild reactions, is as follows:—(1) The first thing is to assure the patient who gets perturbed, that these symptoms will pass away and to give him confidence and courage. (2) To put him to bed or at least compel him to take absolute rest. (3) Give a diaphoretic mixture with 10 grs. of sodii salicylas per dose. (4) For the body pain, powders of aspirin, phenacetin and caffein

citras 2½ grs. each are given. (5) For sleeplessness and restlessness, particularly neuritis, a mixture containing Pot. bromide 15 grs. Chloral hydras 5 to 10 grs. per dose may be given twice or thrice a day. This mixture has an even better effect than the A.P.C. powders. (6) Intravenous injections of 10 c.c. Calcium gluconate 10% are most useful. (7) Injections of 20 c. c. glucose 25% also help a lot and may be given daily. If fever persists, injections of Pot. antimony tart. intravenously in doses of 2 c.c. of 2% solution or Anthiomaline or Fantorin intramuscularly in doses of 2 c.c. may be given—six injections. At the end of this period, a course of vitamin B_{12} injections and iron by mouth will help the convalescence. For anæmia, vitamin B_{12} may be supplemented by injections of liver extract and folic acid. For neuritis one has to rely mostly on Chloral bromide mixture; locally injections of saline and novocaine may be tried, and may be given freely. Morphia may have to be used on occasion and should be given when required. After the acute pains have subsided, Berin and vitamin B_{12}, are useful tonics. For jaundice, small doses of calomel, Livergen or hepatic mixture, intravenous calcium with glucose would be the treatment. For dermatitis, the treatment is conducted as for mild reactions with rest, diaphoretic mixture and locally calamine lotion, lead lotion when there is weeping and later on calamine cream with olive oil and Lassar's paste may be applied. Injections of milk protein may also be tried to hasten recovery. The bowels must be kept free. For eye reactions and for conjunctivitis, Argyrol and sulphanilamide ointment, and for iritis and other eye affections, energetic appropriate treatment must be instituted at once.

After the reactions and toxic effects subside, how long should one wait to resume treatment? What is the line of treatment to follow? The following rules will serve as a guide. (1) All erythematous and red patches must completely subside. (2) There should be no nerve or body pains. (3) Fever should subside. (4) Weakness and anæmia must be got over. (5) Appetite for food must return and the patient must have a sense of well-being. This may take anything from six to twelve weeks. After this, injections of sulphetrone commencing with small doses of ½ c.c. and gradually increased, keeping an eye on the hæmoglobin index, and watching for any further reactions, may be given once and later twice a week.

I would also like to mention that a few minor surgical ailments such as abscess, extraction of teeth etc. can be treated in private clinics. The use of penicillin and soluseptasine is extremely useful in all infections as it minimises time and reduces the suffering of the patients.

The above is an outline of the treatment for leprosy and some of its common complications. Given proper facilities for laboratory examination in the mofussil, it should not be difficult for

practitioners to undertake the treatment of cases of leprosy. Provision should however, be made for all patients suffering from advanced lepromatous leprosy and neural cases with deformities or requiring other surgical treatment to be admitted into the Leprosy hospitals in the area. Given the above facilities, the treatment of cases of leprosy by private practitioners, particularly in the mofussil, would not only give relief to many a sufferer from the disease but would also help to build up a scheme for the effective control and relief of leprosy in the districts.

Para-Acetamidobenzaldehyde Thiosemicarbazone in the Treatment of Leprosy

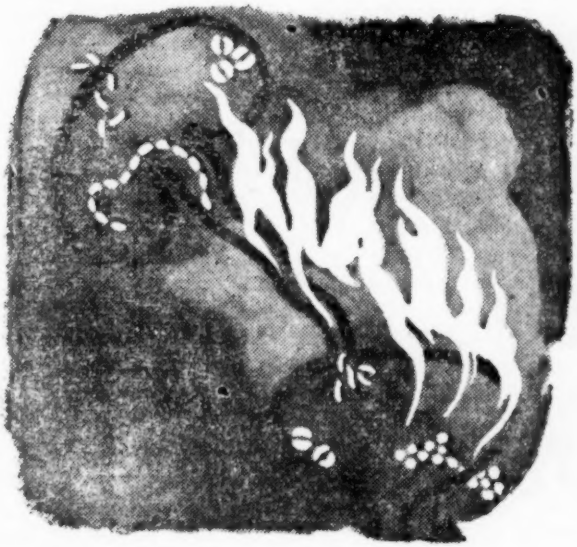
Patients in Nigeria suffering from leprosy were treated with *p*-acetamidobenzaldehyde thiosemicarbazone (T.B.I. "berculon A"). Seventy one patients were treated for 5 to 13 months (average 8½). The dose used was 50 mg. daily for 3 or 4 days, then 50 mg. twice daily for 3 or 4 days and thereafter 50 mg. in the morning and 100 mg. in the evening.

Serious toxic effects were seen in only one of the 71 patients who developed acute agranulocytosis. He had been treated for 4 weeks when he complained of malaise and pain in the teeth. Later in the day his temperature was 103° F.; he was ill and toxic, and the gums, tonsils, fauces and nail beds of the toes were acutely inflamed; the polymorphonuclear leucocyte count was 400 per c.mm. (lymphocytes 2700 per c.mm.). He was treated with procaine penicillin in oil, 1,000,000 units daily and recovered. Treatment of this patient was continued with sulphone without incident. In the other 70 cases, only minor toxic effects were seen such as anorexia, epigastric discomfort (in 12) and fever and rash (in 2). There were also exacerbations of the leprous inflammations.

The patients could be divided into 3 groups:—Group 1 consisted of 30 lepromatous cases, many of them severe. A lepromatous reaction occurred 8 times, neuritis once and iritis once (7 in all). In all these cases, there was clinical and bacteriological improvement. Group 2 consisted of 28 cases of the active tuberculoid type. No complications occurred. There was slow clinical improvement in all cases; (bacteriologically the cases were already negative). The 3rd group consisted of 13 lepromatous cases previously treated with sulphone, to which reactions had occurred. There were numerous lepromatous reactions but these were fewer than after sulphone therapy. Clinical improvement occurred in 12 of these cases and bacteriological improvement in 5 cases.

Compared with sulphone, T.B.I. does not produce allergy to itself, but it is less cheap and less simple to give; the results are similar to those of sulphone. It is concluded that T.B.I. may be a useful alternative to sulphone in patients in whom complications are many and severe. It should be restricted to patients kept under supervision in a hospital.

A further 55 patients have since been treated and there have been no more severe toxic effects or complications.—(Lowe. J., *Lancet*, pp. 436-439, 1-3-1952). (*Abst. World Med.*, Aug. 1952).



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THE PRESENT STATUS OF SULPHONE THERAPY IN LEPROSY

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FROM early times, Chaulmoogra or Hydnocarpus was known to have beneficial effects and was resorted to, as soon as the disease manifested itself. Later, the introduction of intradermal injections brought better results. But there was little security, as far too many who were discharged from a sanatorium free of the disease had relapses which brought them back to the Home or Sanatorium for further treatment. Intradermal and intra-muscular injections are very painful experiences and it is not true that every prick in a leprous skin is painless. The patient certainly pays heavily for any results obtained and it is rather remarkable that every patient goes through the ordeal so cheerfully and so hopefully. No doctor has used the word ' cure ' and the best that could be hoped for is to become an arrested case with the possibility that no relapse would follow. With the advent of the sulphone and its derivatives, dramatic and remarkably good results have been obtained and there is now great hope for the more advanced cases which in the hydnocarpus age were thought to be practically hopeless cases.

Since the discovery of the anti-bacterial properties of the sulphonamides by Domagk in 1935, a new era was ushered in and attention was focused to find a suitable drug for the treatment of microbacterial diseases, namely, Tuberculosis and Leprosy. The discovery of prontosil and its activity against many of the acute coccal diseases, and the findings that the action of Prontosil was due to its sulphonamide radical, and that such compounds like Prontosil might show similar bacteriostatic properties led Buttle *et al* to focus their attention on Diamino-diphenyl-sulphone which was synthesised many years ago by Fromm and Wittmann in 1908. This substance was found to be very effective, more effective than the sulphonamides. But experimental work in 1937 showed that it had a high degree of toxicity. Naturally therefore, having found this parent substance Diamino-diphenyl-sulphone to be very active in experimental tuberculosis, and having learnt that this is very toxic, attempts were next made to produce substitution compounds of this parent substance, in order to render it less toxic and make it available for use in tuberculosis.

About 1940, Feldman *et al* commenced to use a derivative prepared in 1935 by Parke Davis & Co., Promin which is a di-substituted sulphone of the chemical formula N–N–di–dextrose sulphonate with a molecular weight of 248 and DDS content of 31·8%. This drug when tried in experimental tuberculosis in guinea-pigs, was found to have very good effect. This led to a further search for drugs of this group, not only for the treatment of tuberculosis

but also for leprosy and other serious micro-bacterial infections which afflicted mankind.

Hence about the year 1936 sulphetrone with a chemical formula of Cinnamaldehyde bisulphite derivative of DDS with a molecular weight of 892·5 and DDS content of 27·8% was prepared by Burroughs Wellcome & Co. It was followed soon after in 1938 by the Abbott Laboratories who put on the market diasone which is a formaldehyde sulphoxylate derivative of DDS with a molecular weight of 448 and D.D.S. content of 55·4%. Parke Davis followed it up with diamidin which is similar to diasone and promizole which is a thiazole derivative of DDS having a molecular weight of 255 and DDS content of 97·5%, and promacetin which is a sodium diamino-diphenyl-sulphone acetyl sulphonamide with a molecular weight of 390, and DDS content of 63·6%. This was later followed up by the Swiss Company Cilag who put out sulphone-cilag a monoacetyl ester of DDS with a molecular weight 328·8 and DDS content of 75·7 which differed from the above di-substituted sulphones in being a mono-substituted sulphone. Again in 1946 Domagk of Bayer Laboratories, Germany, produced Thiosemicarbazone which has an open chain-like arrangement of nitrogen and sulphur atoms in the Thiosemicarbazone which is responsible for its activity and this substituted with aceto-aldehyde in the para-position further augments its activity; hence we get the formula *p*-acetamidobenzaldehyde thiosemicarbazone with a molecular weight of 236·68.

Many of the sulphone preparations which are at present available for the treatment of leprosy have been enumerated above. As early as 1940, when sulphetrone was tried in experimental leprosy, glowing tributes were published in some journals about the efficacy of this drug in the treatment of leprosy at the Leprosarium at Carville (U.S.) and it was thought that at last the remedy, specific for leprosy had been found. Of course, after years of experimentation and trial we have come to learn that we have not yet got in our hands a drug which could be called *the specific* for leprosy. No doubt we have entered the era of chemotherapy in leprosy. But chemotherapy is a system of treatment by which the medication with a drug is undertaken with a view to bringing about a cure within a reasonable time and making the present treatments obsolete. But that hope is still far away.

Absorption, excretion and mode of action of sulphones.—DDS was found to have marked anti-bacterial action against gram-positive organisms *in vitro* and in animals especially of the streptococcal group. It was also found to exert a powerful action over the micro-bacterium tuberculosis in animals. Hence, as already stated, attempts were made by Buttle *et al* to use it in doses of 1 to 2 gm. per day, which gave rise to serious toxic effects and this led to the search for less toxic derivatives. Attention was focused

in using the disubstituted compounds for the treatment of leprosy, and hence to day we have these products available as the standard treatment in leprosy. But we find these complex sulphones expensive to use on a large scale and for widespread leprosy treatment in undeveloped and poor countries where leprosy is endemic. A cheaper sulphone was urgently needed. In the search for such a substance which would satisfy conditions such as administrative and pharmaceutical simplicity, and cheapness for use in mass therapy, workers in Britain, France, Brazil and India saw reason to believe that since substituted compounds acted by virtue of their free DDS content, DDS itself might be used in much smaller dosage, would be cheap and also prove a powerful therapeutic agent in the treatment of leprosy.

McEwen *et al* in 1941, used DDS in veterinary medicine for the treatment of mastitis in cattle. DDS was used in a 20% suspension. Cochrane *et al* in India, Lowe and Smith in Nigeria and Molesworth in Malaya were the first to take the parent substance for trial in the treatment of leprosy. Cochrane, it must be observed here, was the first in the field to use DDS by means of injections; and unfortunately the dosage being too big, toxic symptoms were rather serious though the results were very promising. He was quickly followed by Molesworth and Narayanaswami who followed Cochrane's example but gave this drug in smaller doses with very good results. Simultaneously, Lowe gave this orally in low and very gradually increasing doses without any serious toxic effects and obtained results as good as complex sulphones, and at a fraction of the cost. Smith also made pharmaceutical studies of the different sulphones and much has been learned regarding absorption, excretion and therapeutic values of many of the sulphones. At the same time Floch and Destombes also reported from French Guinea, on the efficacy of DDS in small doses using both the oral and parenteral routes. At the same time D'Souza Lyma in Brazil using DDS orally, reported results similar to those of the British and French workers. With this fund of knowledge relating to the proper use of DDS, workers are again turning their attention to the treatment of tuberculosis with DDS and it has been noted that streptomycin-resistant cases of tuberculosis when treated with DDS respond to streptomycin far better than when treated with streptomycin alone. It has also been noted that PAS-resistant cases also respond better when combined with DDS medication.

While studying the action of sulphones and their derivatives it has been found by certain workers that many of the di-substituted sulphones, theoretically having a closed chain, become blocked and may be considered inactive, but it is believed that because of their instability the action of many of these complex sulphones may be explained. This is probably the crux of the whole problem. Complex sulphones, it is believed, remain in the body in

their original complex form, while some are degraded to DDS and possibly other derivatives. But it is impossible to say how much or in what percentage they are so broken up. DDS is readily soluble in organic solvents such as benzene ethyl acetate, but is practically insoluble in water, whereas the di-substituted products are readily soluble in water but insoluble in organic solvents. From this knowledge it has been possible to work out the DDS content of the circulating blood while under the administration of the di-substituted sulphones.

Regarding the instability of the di-substituted sulphones *in vitro* it has been found that especially with regard to the preparation of sulphetrone as a 50% solution, Brownlee has reported that when the solution is neutral or slightly alkaline, a 50% solution can be autoclaved, and further that sulphetrone cannot be obtained as a pure substance and that 6% of it was not sulphetrone in the chemical sense. Bearing these points in mind and also the facts that sulphetrone and promin are both very unstable in concentrations below 5% and that they have been known to precipitate as DDS, and the same hydrolysis may take place if a little acid is added, it may be stated that (1) freshly prepared solutions of sulphetrone contain a considerable trace of DDS; (2) sulphetrone in solution is unstable and is easily degraded to DDS and that the conditions which favour this degradation are high dilution, temperature, and acidity; and (3) that the maxim of this is probably one of hydrolysis and that the greater stability of concentrated solutions, namely 50% is due to lack of H. ions to hydrolise much of the large amount of sulphetrone precipitant. This influence in instability explains the chemotherapeutic activity *in vitro*. While studying the activity of these di-substituted sulphones by the oral and the parenteral routes it may be said that by the oral route the acid hydrolysis that takes place in the stomach is responsible for the liberation of DDS, but by the parenteral route it is not quite clear how these di-substituted sulphones break up, unless it be explained that the body fluids combine with these substances to produce water-soluble mono-substituted sulphones which are absorbed to produce the therapeutic effect.

Further, the influence of this instability on the chemotherapeutic activity *in vitro* of the different di-substituted sulphones has been investigated by Boyer and his co-workers who studied the activity of the several sulphones against Proteus X. 4. Before and after autoclaving both strong and weak solutions they found that in a 50% solution the activity of sulphetrone was increased only 5 times after autoclaving, but in a 5% solution the increase was twenty-fold. They attributed the increase of activity in the complex sulphones on autoclaving, to the liberation of DDS by hydrolysis.

Therefore, there is no reason whatsoever to suppose that di-substituted sulphones act by virtue of their whole molecule, either

in vitro or *in vivo* without giving off DDS. By applying this principle, the early experience of Feldman *et al* who found oral Promin was more toxic than injected Promin can be easily explained. Further experimentation in animals of the study of di-substituted sulphones given both orally and parenterally to mice and dogs and of estimating the body concentrations of both DDS and complex sulphones it has been found that on administration of complex sulphones by either methods, DDS was found in the blood, but that oral administration produced considerably higher levels than injections. This was attributed to acid hydrolysis in the stomach. But the DDS level found by the injection route cannot be well explained except on the assumption that these complex sulphones do contain considerable impurity in the form of DDS to start with.

While studying the effect of oral administration and injection of the di-substituted sulphones in man, it is doubtful whether the amount of DDS level produced is sufficient to explain fully the therapeutic effect. Sulphetrone given by mouth in doses of 3 to 6 grams a day produces a free DDS blood level of 0·2 to 0·5 mg. per cent. Diasone in its usual dose of 0·9 to 1·8 gm. per day gives a DDS blood level from 0·2 to 0·6 mg. per cent. It will be noticed that sulphetrone produces less DDS than diasone, hence the variation in the oral dosages. The question whether the blood DDS is high enough to explain the therapeutic action of these sulphones can be answered in the affirmative. It is believed the therapeutic blood level of DDS in leprosy is certainly as low as 0·2 mg. per cent and may possibly be still lower. The blood DDS levels produced by injections of di-substituted sulphones are as a rule very low. Actually they may not be more than the explainable amount present as traces of DDS as impurity in the sulphones injected. If the therapeutic effect of such injections is due to the DDS content, the therapeutic dose of DDS must be very small. A therapeutic DDS level of 0·2 mg. per cent has been said to be therapeutically active, but it is doubtful whether levels of 0·1 mg. per cent such as are produced by sulphetrone injections are enough to explain the therapeutic action of these injections. It is possible that other factors contribute to this action. It is possible that injections of di-substituted sulphones are therapeutically active partly because of impurities present before the injections, and after injection mono-substituted sulphones may be produced and these are responsible for the action. This aspect needs investigation.

Absorption of orally administered DDS is almost complete. About 85% of the sulphone given appears in the urine later. Excretion is mainly by the kidneys. Considerable amounts may be rejected in the fluid excretion of the body including the sweat, saliva, tears, etc. Excretion by the kidneys is a slow process, and therefore, it has been noticed that in a patient receiving ordinary doses of DDS, sulphone is found in the blood in appreciable

amounts, often for several weeks, and may be detected in the urine for even longer periods. When this excretion by the kidneys was examined for its DDS content, it was found that the sulphone excreted was no longer in the form of free DDS, but in a water-soluble form. It is a matter of conjecture whether this water-soluble form which is found both in the circulating blood and in the urine is mono-substituted sulphone, which as we know, is therapeutically more active. During the early period of experimentation with this drug, great stress was laid on its concentration and storage in the body tissues, especially by Cochrane who considered that sulphones including DDS are concentrated and retained in the skin. But this has not been supported by other workers. However, the only tissues that concentrate sulphones in any appreciable quantity are the kidneys and the liver which excrete sulphones. It is this complete absorption and slow elimination of DDS that makes it such an economical therapeutic agent and hence high dosages are dangerous because of the cumulative effects produced.

It has been experienced that acute toxicity is seen by blood levels of 2 mg. per cent and above. To produce this level about 2000 mg. of DDS is required per week. Levels between 1 mg. and 2 mg. per cent produce negligible toxic effects. Therefore, a safe blood level lies as already mentioned, between 0·2 and 1 mg. per cent, and averaging about 0·5 mg. per cent. If this is conceded, it will be found, that there is a considerable margin between the toxic and the therapeutic blood levels, and it may be deduced that the therapeutic doses of DDS should range between 300 mg. to 1200 mg. a week without producing toxic effects.

Many workers on leprosy such as Lowe and Smith of Nigeria, Flock and Distombes of French Guinea, Rist and Cottet of France, D'Souza Lyma of South America, Muir, Cochrane, Dharmendra of India now agree that a dosage not more than 200 mg. per day is a safe and effective dose. Molesworth from Malaya has used weekly injections upto 500 mg. without toxic effects. A dose of 500 milligrams is well tolerated for treatment twice a week and Lowe subscribes to this view that twice-weekly-treatment now appears preferable to daily treatment though a weekly dose of 600 mg. is also being investigated. From the concensus of opinion, it is now found that 200 mg. a day or twice-weekly doses rising slowly to 400 mg. or weekly doses rising slowly to 500 or 600 mg. are equally well tolerated and produce good response.

Action of sulphone drugs.—It is now well known that all sulphones have the property of causing the micro-bacterium lepræ to break up into granules. Granular forms are however, seen not only in sulphone therapy, but also under hydnocarpus therapy as well and during the natural evolution and resolution of the disease. In an advanced lepromatous case, enormous numbers of bacilli can be demonstrated from skin smears and to see after 18 months or

two years the almost complete disappearance or perhaps some acid fast dust is most striking. Whether these fragmented forms of the bacilli are viable or not is controversial. It has been found by Khanolkar and Cochrane that when the bacilli disappear from the skin, granular forms can still be demonstrated in the small subcutaneous nerves and in the muscular nerve endings, and they therefore, suggested that these may act as reservoirs of infection from which a recrudescence of the disease is possible. It is probable that these forms are inactive and that a patient may be non-infective when a few granules are left. It has also been observed that lepra-reactions may occur even after the bacilli have almost completely disappeared from the skin Therefore, it may be presumed that acid fast granules are still viable forms of the bacillus and as Cochrane concludes : (1) although there is at present no definite evidence that the granular forms of bacilli are dead, in all probability they represent a stage of relative inactivity and are therefore, much less capable of transmitting the infection ; (2) Sulphones prepare the tissues of the body for the alteration of the bacilli into granular forms and thus enable the microphages to deal with them more easily ; and (3) in view of these findings it may be well to continue maintenance doses of sulphones for a considerable time after clinical cure and possibly throughout the life of the patient.

Even though with the advent of sulphones, there is greater hope of success in the treatment of leprosy, the limitations are by no means small. It has been noticed that the sulphone therapy is a long term programme, and the time required for the complete elimination of the lepra-bacilli from the skin and nerve varies anywhere from 2 to 5 years and even more in some of the very advanced cases. As has been stated above, even though the bacilli may disappear completely from the skin they may still persist in the nerves. It may be argued that because a lepromatous case is given sulphone therapy early it will become inactive more quickly than one which is more advanced. It has been noticed that marked clinical and bacteriological improvement in moderately advanced and advanced lepromatous cases has been noticed, whereas in the early cases the progress has been very slow. Another and more serious drawback of sulphone treatment is the tendency to relapse after clinical arrest of the disease. This question will have to be confronted as the years go by, and hence as suggested by Cochrane, the drug may have to be continued possibly throughout the life of the patient.

The treatment of patients with sulphones may be advantageous for a quick turn-over of patients in leprosy institutions and for minimising infectivity in the out-patients, for the reason that these drugs are able to produce granular forms in a shorter period of time.

It is needless to refer at this stage to universal beneficial effects of sulphone treatment in all lepromatous cases which are most manifest in the advanced cases with extensive infiltration of the skin, nodulation, ulceration with complicating lesions of the eyes, nose and throat. With the very commencement of sulphone therapy, it is noticed that further progress of the disease is checked and there is gradual regression, slow but steady, towards marked clinical and general improvement of the patient. Both subjectively and objectively the improvement is most striking after about six months, on an average. The early signs of improvement are healing of leprotic ulcers, and of ulcerations in the nose and throat. But from the study of sulphones through the years, a general all round improvement both clinical and bacteriological is seen after about $2\frac{1}{2}$ to 3 years.

From our experience in the therapeutic assessment of cases in the Lady Willingdon Leprosy Sanatorium, it is being increasingly felt that whatever type of sulphone is used and in whatever dosage, it is the period of administration of the drug over 3 to 4 years that strikes one as the chief factor in the treatment of leprosy, and further that the various types of sulphones administered in the various forms have produced almost the same negative rate in almost the same period of time. Again from the economic point of view, the basic sulphone in whatever form used is the cheapest.

Toxicity.—Apart from the usual more serious toxic signs such as nausea, vomiting, jaundice and psychosis which are due to inadvertent and heavy dosages of sulphones, a condition described by Wolcott as erythema nodosum leprosum has been noticed. This syndrome may be likened to Herxheimer reaction and presents an acute response to the rapid multiplication and breakdown of the micro-bacterium lepræ giving rise to high fever, erythematous nodules and erythema-like lesions. The condition is on the whole favourable and calls only for stoppage of the drug for the time. When the drug has been stopped, it should be re-started with caution. This condition is not to be confused with the extension of activity of the disease. This condition is more liable to be provoked by the administration of the parent sulphone than by the di-substituted sulphones. The most common toxic sign is a decrease in the blood-cell-count with a fall in hæmoglobin percentage. And with either the parent substance or the di-substituted sulphone, its tendency to produce anæmia may be serious and calls for either stoppage of the drug for some time or concomitant treatment with iron and vitamins. Another distressing condition which is seen in both sulphone and sulphetrone treatments is a drug-dermatitis, which requires very careful treatment, though it does usually respond to antihistaminics. This allergic condition may be eliminated by gradual desensitisation of the patient to the drug by administering minute doses, gradually working up to the optimum level.

Neuritis.—Neuritis is a distressing symptom which sometimes requires careful handling and has been observed in a small percentage of sulphone-treated cases. This condition which may have been basically latent, is probably provoked to potency and presents itself as one of the complications of sulphone medication, particularly seen in sulphetrone administration. It is interesting to note that this is not complained of, before commencement of treatment, but arises during the course of treatment at a stage when the bacteriological index has fallen to 0·5 and below. At times it is fairly acute necessitating surgical interference. There seems to be no gross thickening of nerves except an œdematous swelling and the sheath on stripping, varies in thickness being occasionally adherent. The results of the operation have been most gratifying.

Sulphone therapy in relation to the type of case.—Now that the type of sulphone which will conform to cheapness of administration has been decided on, it may be said that every active lepromatous case should receive this therapy. Lowe (1950) has adovcated that all tuberculoid cases should be given sulphone therapy. This has been investigated, and the claim can be endorsed that all tuberculoid cases should be given sulphones. Further, it has also been found that atypical lepromas respond very well to sulphones. Of course, there are the indolent types which may be styled as progressive tuberculoid lesions or atypical lesions which will not respond as dramatically as the more active lesions. In such cases, it has been our practice, when no marked improvement is seen after 8 months, to give also intradermal injections of ethyl esters of hydnocarpus oil in addition. In the hypopigmented macules of neural leprosy there is no means of assessing the efficacy of these drugs and if one is certain that the neural lesions are progressive and active (simple neural type: Cairo), it will not be wrong to put these cases on sulphone therapy. Though experience has not been conclusive, it may be said that the administration of sulphone in maculo-anæsthetic cases has proved to be of benefit in checking the activity and spread of the lesions. Here again it will be noticed that sulphone therapy will not clear the hypo-pigmented patches which may remain as resolved lesions either of the tuberculoid, atypical or early macular lepromatous leprosy and this calls for intradermal injections of ethyl esters of hydnocarpus oil. Apart from this, sulphone therapy should be used as already mentioned in all bacteriologically positive cases and more so every advanced lepromatous case requires sulphone treatment (Cochrane).

While reviewing the drug of choice for out-patient and in-patient therapy it may be argued that considering the cost of treatment and administrative simplicity for mass treatment, DDS should be used either by the parenteral or the oral route. With due consideration to the toxicity of this parent drug for mass administration and for better control either in an institution or for out-patient

therapy the parenteral route is preferable when dealing with the cross-section of society which still feels to a large extent that a drug administered by the needle produces better effects than the homœopathic doses which have to be advocated. Hitherto in an institution like the Lady Willingdon Leprosy Sanatorium, sulphetrone as a 50% solution has been used, for sulphetrone by the parenteral route produces the desired beneficial result, with the least toxic symptoms. While comparing this with the administration of DDS in a 25% suspension and in the dosage which will be effective for optimum therapeutic effect, the latter is decidedly cheaper than the former and there is no reason why this should not be universally adopted for mass treatment of leprosy. People from far and near come once a week for out-patient treatment; parenteral administration of DDS is, to my mind, the ideal, as a single dose of 600 mg. per week gives most satisfactory results.

Conclusion.—Absolute success of modern therapy in leprosy is still awaiting solution. For, as has been already stated, we are still very far from having a drug which could be called the specific for leprosy, though we have now on our hands only bacteriostatic drugs. The leprosy problem still awaits solution for a similar or better therapeutic drug; and having found such a drug we should not be unmindful of the need for the study of physiotherapy and orthopædic measures in leprosy so that deformity, threatened or actual, may be alleviated.

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Chemotherapy of Tuberculosis and Leprosy with B. 283: a Phenazine Pigment

This drug is the hydrochloride of 2-anilino-3 imino-5 phenyl phenozine. In Proskauer and Beck's synthetic medium, it inhibited M. tuberculosis for 6 weeks at a dilution of 5×10^6. Dr. Barnes of Nigeria reported the results of treatment of lepromatious leprosy with B. 283. He stated that resolution of nodules occurred without six months in six of ten cases treated. Anæsthesia was slower to respond than the nodules. The number of bacilli obtained in smears from the nose and skin became fewer in these six cases and in one case bacilli disappeared. Dr. Barnes considered B. 283 to be more active against leprosy than any other drugs used in Ogoja (Nigeria) including chaulmoogra oil, sulphetrone, promin, and thiosemicarbazone. Evidence of mild liver damage was observed in some patients, but this cleared up on withdrawing the drug. He found it best to start with 250 mg. daily.—(*Leprosy in India*, Jan. 1953, From *L. W. Memorial* 3: 1952).

RECENT ADVANCES IN THE TREATMENT OF LEPROSY*

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THE old notion that leprosy was incurable is fast losing ground thanks to the modern advances in treatment; as a result we could now arrest the disease, make the sufferers symptom-free, the infective persons non-infective, and to some extent even cure the sufferer and by removing the stigma attaching to him make him fit to move freely in society.

The treatment of leprosy has made very rapid progress during the past 15 years. The old treatment with hydnocarpus oil and its derivatives, has still its advantages. Good results have been noticed in our institution after prolonged and continuous treatment. In leprosy, unlike other diseases the treatment is a very prolonged and tedious one which generally discourages the patient from taking the full course of treatment. So, those interested in the treatment of leprosy always anxiously wait for further advances and developments. Workers in different countries have tried various remedies. After persisting with sulphone treatment for leprosy, enthusiastic workers in America and in other countries have succeeded in establishing the sulphone group of drugs as the treatment of choice for leprosy. So different preparations of sulphone drugs have come into the market and we had a chance of getting and trying Diasone from Abbotts Laboratories, although it was very costly at that time. Under the expert guidance of Dr. Cochrane, half a dozen cases of the advanced lepromatous type were selected for experiment and placed on Diasone treatment.

Dose:—To start with we gave five 0·3 gm. tablets orally every day (6 days in the week). All the six cases were fairly advanced ones with extensive lesions and ulcers on the skin and nose and involvement of larynx with hoarseness of voice in two cases, and involvement of the eye in one case. The average duration of treatment was 2 years.

Clinical results:—Healing of ulcers on the skin and nose was seen. Clearing up of the lesions in the nose and throat was noticed. Leprous iritis and eye reactions also responded well. The lesions in the larynx gradually healed up and hoarseness of voice diminished greatly. Nodulations and thick infiltration areas in the skin began to shrink. Marked all round improvement was noticed in all these cases. One case which had been deteriorating with hydnocarpus oil treatment, showed very marked improvement after treatment with Diasone.

* Based on the results of experimental work carried out in the Vadathorasalur Leprosy Home in Madras State.

Bacteriological results : —There was marked improvement in all the cases getting Diasone. In two cases the bacteriological index came down from 3·5 to 0·5 within a period of 18 months ; this was not so when hydnocarpus oil was used.

Toxic effects of Diasone :—The most common toxic effects are the production of anæmia and a feeling of fatigue, general weakness and giddiness, gastric disturbances, such as nausea and vomiting ; mental disturbance was also noticed in two cases and dermatitis in three. While we were experimenting with Diasone, a sulphetrone preparation came into the Indian market and was widely welcomed as an up-do-date therapeutic agent for the treatment of leprosy and has gained importance in the treatment of leprosy during the last four or five years. We started treating some of our cases with a 50% aqueous solution of sulphetrone parenterally twice a week in all lepromatous cases, under Dr. Cochrane's guidance.

Dose :—Three groups were taken up for treatment : Each group consisted of 25 cases. One group was given 3·5 c.c. ; one was given 5 c.c. and the third was given 7 c.c. As we gave these different doses for several months we noticed that the 3·5 c.c. group, showed the same degree of improvement as the 5 c.c. and 7 c.c. groups. So, instead of wasting sulphetrone we redivided the groups into A, B and C, giving them 3·5 c.c., 2 c.c. and 1 c.c. respectively, twice a week in order to ascertain the minimum effective dose needed to obtain adequate benefit, without serious toxic side-effects.

Clinical improvement :—The healing of intractable ulcers, clearing of the nasal passage and other affected mucous membranes and arrest of the progress of the disease in the eyes were noted. Nodules and thickenings of the skin began to flatten out. In a few less advanced cases, this improvement was rapid and early cases became negative within a few months. These effects appeared in a remarkably short time, an observation which was not noted with hydnocarpus oil treatment. It had taken a little longer time with diasone.

Bacteriological improvement :—There was a remarkable reduction in the bacteriological index and there was a marked and progressive change in the structure of the bacilli, from fragments through rough and fine granular stages to that of a fine dust.

In order to determine its value in other than lepromatous cases, we selected a few tuberculoid and indeterminate cases and treated them with sulphetrone. These cases also showed marked changes, but with a slight residual hypo-pigmentation. After a few intradermal injections of an equal quantity of the ethyl esters of olei hydnocarp, with hydnocarpus oil, these patches disappeared leaving only the scars.

Our experience shows that most of the patients under sulphetrone treatment improve considerably even without iron and yeast and we seldom use iron except for Diasone cases. In fact, we have noticed the low Hb. index rise up even without iron as the general health of the patients improved. Sulphetrone treatment is suspended in cases whose Hb. index is 50% and below; they are first treated with iron and yeast and sulphetrone is then continued when Hb. rises above 50%.

Toxic symptoms:—No marked toxic symptoms were noticed except the lepra reactions which, in a few cases, were very severe with the appearance of big rose-spot nodules on the face with ulceration, multiple subcutaneous nodules all over with severe pain on touching them, pain all over the joints with a hectic temperature. In a few cases occasional headache and itching were noticed, and these symptoms disappeared when the patients were given saline purgatives *e.g.*, mag. sulph.

Preparation of sulphetrone:—We follow the standard method of preparation. We store rain water, as much as possible, and use it instead of distilled water; no untoward effect has so far been noticed and the pain of the injection is well tolerated.

D. D. S.:—As a result of the experience and knowledge gained by leprologists working with sulphone groups, Diaminodiphenyl sulphone (DDS) came into use. This drug was once thought to be highly toxic and expensive and was not therefore, used extensively in leprosy cases. Recent experiments by several workers have made it possible for this drug to be widely used by all without fear of toxicity or high cost. Both in India and abroad, leprologists have since been using it freely for leprosy cases. So we also started using this drug because of its low cost and ease of administration. Thirty cases of all types of the disease of different ages and sexes were selected for this experiment. Six identical cases treated with hydnocarpus oil, served as control.

Method of administration of DDS:—Because of the ease of administration and the extraordinarily high degree of absorption from the gut, the oral route is generally preferred.

Dose:—50 mg. of the drug in tablet form twice a week, increased every fortnight by 50 mg, until 300 mg. were taken twice a week *i.e.*, 600 mg. weekly. After a couple of months on this regimen, the dose is increased to 400 mg. twice a week *i.e.*, 800 mg. We gave this higher dose *i.e.*, 800 mg. per week for a few months but reduced it to 600 mg. per week, as a few weak patients experienced slight *toxic symptoms*, like nausea, dermatitis, lepra-reaction and anæmia. Rest with yeast therapy brought such cases to normal when 300 mg. twice a week were continued without any untoward symptoms.

TABLE

Statement showing the Clinical Improvement

No.	Age	Type	Dose in c.c.	Length of treatment in months	Total dose in gms.	Date of commencement	Condition before treatment
A1	18	L_2	3·5	38	577·5	1-8-'49	Dif. leprom. lep. with infilt. of face forehead and ears; loss of eye brows; no neural deformity.
A3	19	L_3	3·5	38	570	Do.	Infilt. leprom. lep., face with large succulent coalescing nodules with marked infilt. of nose and ears; loss of eye brows, nasal blockage, no marked throat involvement. No deformity.
A4	33	L_3	3·5	38	570	Do.	Infilt. leproma, eyebrows marked infilt. and lost, ears thickened and pendulous definite leprom. infilt. of the eyes; nose deformed and hoarse voice, laryngeal involvement, slight deformity of hands.
A7	53	L_3	3·5	38	558	Do.	Infilt. leprom, marked nodulation of forehead and chin, loss of eye brows, ears and nose markedly thickened. No marked neural lesions.
A9	27	L_2	3·5	24	323	Do.	Macular leproma with some slight infilt. on body, face and ears infilt. eyes mild scleritis—no deformity.
A10	35	L_3	5 c.c. to 3·5 c.c. (8/6/50)	38	635	Do.	Nodular leprom. nodules more marked on back of arms, ulcerated nodules on right forearm, face, shiny and infilt., small nodules on upper lip, nose and forehead, slight deformity of the right hand.
A12	24	L_3	Do.	38	635	Do.	Advanced leprom; infilt. all over, much debilitated condition. Wt. 62 lbs., infilt. of face, lips swollen, mouth reduced to small circular opening of about 1½" tongue marked with leprous glossitis, eyes affected; particularly left almost blind, nails affected, hands commencing contractures, hoarse voice indicating laryngeal affection.
A18	37	L_3	7 c.c. to 3·5 c.c. (8/6/50)	38	715·75	Do.	Very advanced leprom. lep., dif. infilt. with numerous nodules very advanced lesions on the face which is thickened and infilt. and deformed, lips infilt, tongue marked glossitis, well marked circumscribed leproma on both the eyes invading left eye cornea, pendulous ears, impaired vision, cannot count fingers, hoarse voice, trophic ulcer left foot.
D1	24	N_2	4 c.c.	22	424	3-7-'50	Numerous erythematous and gross infiltration. Macules with raised and well marked edges with loss of sensation, contracture of left hand.

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obtained with Sulphetrone 50% Aqueous Solution.

B. Index.		HB. Index		Reaction mild moderate or severe	Clinical improvement
At commencement	Now	At commencement	Now		
3·18	÷ (negative)	76·0	91·17	None	Complete subsidence of lesions, looks normal.
5·56	0·5	55·0	82·35	Mild	Considerable subsidence of lesions succulent nature of the nodules disappeared. Slight infilt. remaining.
5·5	0·5	67·65	76·47	Do.	Subsidence of the infilt. of face, marked wrinkling owing to resolution.
3·62	0·37	65	85·29	Do.	Very marked clinical improvement, subsidence of lesions of face, eyebrows chin and ears.
2·69	÷	76	91·17	None	Lesions considerably subsided, face appears to be normal. No marked activity of the lesions in general.
4·56	0·5	64·71	79·41	None	Lesions on back considerably subsided and also on face.
5·62	1·25	58·82	76·47	? None	Marked clinical improvement, subsidence of infilt. of face, shine markedly diminished, infilt. around lips subsided marked improvement in the tongue, could open mouth widely. Slight improvement of the eye. Wt. 74 lbs.
5·5	0·75	64·71	70·5	Mild	Very marked clinical imp. considerable reduction of the leproma of the left eye, considerable subsidence of face lesions, could count fingers at 1½", improved vision. There is still slight infilt of faces, forehead and chin
÷	÷	82·35	85·35	None	Almost complete subsidence of infilt. leaving very light hypopigmented macules. Considerable scaling of face lesions. Intradermal injections caused complete disappearance leaving only scar.

TABLE

Statement showing the Clinical Improvement

No.	Age	Type.	Dose in c.c.	Length of treatment in months.	Total dose in gms.	Date of commencement.	Condition before treatment.
B1	7	L_2	2 c.c.	38	274	1-8-49	Multiple shiny slightly infilt. macules over body and extremities, infilt. of nose, cheek, and chin. No neural deformity.
B2	8	L_2	3·5 c.c. to 2 cc. (8-6-50)	38	389	1-8-49	Dif. leprom. lep. face swollen and infilt., ears also; breaking down of glands in left axilla and under chin. No deformity.
B3	10	L_2	2 c.c.	35	299	1-8-49	Multiple shiny lesions over back and buttocks showing slight infilt.; small nodules on the rt. buttock, infilt. of ears, cheek, and chin. No deformity.
B4	12	L_3	3·5 c.c. to 2 c.c. (8-6-50)	38	380	1-8-49	Dif. leprom. lep. marked infilt., nodules on chin, ears and nose. No neural deformity.
B9	15	L_2	3·5 c.c. to 2 c.c. (8-6-50)	28	274	12-12-49	Dif. leprom. lep. with marked infilt. and small nodules over the ears, cheeks and chin, both the arms and back shiny and œdematous with diffuse erythematous skin. No deformity. European type of the disease.
B13	8	L_2	2 c.c.	24	206	25-9-50	Dif. leprom. lep. with infilt. of face and ears, loss of eye brows. No deformity.
C5	32	L_2	1 c.c.	11	49	17-7-50	Dif. leprom. lep. with infilt. of face and forehead. loss of eye brows, nodules on ears and chin, nodules on back, nasal blockage, slight swelling of hands and feet. No deformity.
C6	19	L_3	1 c.c.	19	80	3-7-50	Infilt. leprom. lep. on the body; nodules on ears, forehead and chin, multiple shiny lesions over back and buttock showing infilt. No deformity.
C7	21	L_3	1 c.c	27	89	3-7-50	Very advanced leprom. case, infilt. all over, more marked on face, nodulation of ears, cheeks and chin loss of eye brows, ulcer nose with blockage, harsh breathing. No deformity.
C13	17	L_2	1 c.c.	19	81	5-3-51	Dif. leprom. lep. with infilt. of face, hands and back, loss of eye brows. No deformity.
C19	29	L_3	1 c.c.	24	103	25-9-50	Infilt. leprom. lep. over the body, nodules on ears, cheek, and chin, loss of eye brows; infilt. of lips and nose; multiple shiny lesions on the back, hands buttocks and thighs; Nasal blockage with harsh breathing. Contractures of fingers.

I—(*Contd.*)

obtained with Sulphetrone 50% Aqueous Solution.

B. Index.		HB. Index.		Reaction mild moderate or severe	Clinical improvement.
At commencement.	Now	At commencement.	Now.		
3·68	0·5	64·71	73·53	Severe	Marked subsidence of shininess and infilt. of face. Looks almost normal.
4·12	0·25	61·76	73·53	Mild	Considerable subsidence of lesions of face, swelling disappeared, suppurating glands healed completely; appears to be normal.
3·87	÷	70·59	79·41	None	Complete subsidence of lesions, looks normal.
4·06	0·37	76·47	79·41	Mild	Considerable subsidence of lesions on the chin, ears and nose.
2·62	÷	75·0	85·29	None	Complete disappearance of lesions; looks normal.
2·62	÷	70·59	79·51	None	Complete subsidence of infilt. of face and ears. Normal appearance.
3·5	0·25	79·41	82·25	None	Remarkable imp.; slight infilt. remaining.
3·62	0·68	70·59	85·29	None	Marked appreciable imp., subsidence of lesions of face; some wrinkling. There is still some infilt. The patient appears to have improved considerably.
5·68	1·0	70·59	79·41	Severe	Considerable imp. of lesions of face. Infilt and œdema have subsided There is still infilt. of back and arm with a few subcutaneous nodules.
3·12	÷	73·53	85·29	Mild	Disappearance of lesions all over body. Looks normal.
3·81	1·0	58·82	76·47	None	Considerable subsidence of infilt. of face; œdema has subsided. Marked improvement is noticed. Some infilt. of forehead, arms, buttocks and thighs remaining.

TABLE

Statement showing the Clinical Improvement

No.	Age	Type.	Dose in mg. per week.	Length of treatment in months.	Total Dose in gms.	Date of commencement.	Condition before treatment.
1	27	L_2	600	14	38·2	2-8-51	Dif. infilt. leprom. lep., œdema of the whole body.
2	17	L_1	600	14	38·2	2-8-51	Slightly dif. infilt. leprom. lep. with shining skin, bad ulcers on the legs; scabies all over. No deformity.
3	26	L_2	600	14	38·2	2-8-51	Dif. leprom. lep. with marked infilt. nodules on chin, ears and nose; multiple shiny lesions on back; deformity of the right hand.
4	20	L_2	600	14	38·2	2-8-51	Dif. infilt. leprom. lep. with multiple shiny lesions of the back, nodules on ears and chin, infilt. of face; no neural deformity.
8	46	L_1	600	14	38·2	2-8-51	Thick, dif. multiple shiny infilt. macules all over body. No neural deformity.
11	2	L_1 N_1	600	14	38·2	2-8-51	Dif. infilt. with multiple shiny macules all over, more marked on back. Slight infilt. of the face; slight contractures of the fingers of hands.
13	16	L_2	600	14	38·2	2-8-51	Dif. leprom. lep. with infilt. face nodules on ears, cheeks and chin, loss of eye lesions; no neural deformity.
15	8	N_1	400	14	29·8	2-8-51	A few erythematous and infilt. macules with raised and well-marked edges with loss of sensation on face and hands.
17	13	L_2	400	14	29·8	2-8-51	Multiple shiny slight infilt macules over body, more on the back. Infilt. of ears, cheeks and chin. No neural deformity.
18	25	L_3	600	8	18·8	25-1-51	Dif. leprom. lep. with infilt. of face, ears and forehead, formation of nodules all over, more on ears and nose. No neural deformity.
21	13	L_2	400	6	6·6	17-4-52	Dif. thick infilt. multiple macules all over; infilt. face, forehead and chin, nodules on ears and chin.

Clinical improvement:—The nature and degree of improvement with this drug were found to correlate with the type, the stage and the extent of the disease before beginning treatment. Tuberculoid cases seemed to recover very rapidly. The early lepromatous cases also responded well. The advanced lepromatous cases showed clinical improvement in the first few months of treatment. The atypical or indeterminate cases took a longer time, though we

II

obtained with D.D.S.

B. Index.		H. B. Index%.		Reaction mild moderate or severe.	Clinical improvement.
At commencement.	Now	At commencement.	Now.		
1·62	0·12	73·53	85·29	None.	Marked clinical improvement. Looks almost normal.
1·0	÷	82·35	85·29	Do.	Complete disappearance of the infilt. ulcers healed up. Looks normal.
1·75	0·75	79·41	82·35	Do.	Marked clinical imp. There is still slight infilt.; Sc nodules and shiny appearance.
4·18	0·75	67·65	91·17	Do.	Marked appreciable imp., considerable disappearance of the infilt. and shiny lesions and also nodules on ears and chin. Still slight infiltration.
2·5	÷	82·35	82·35	Do.	Complete disappearance of the infilt. shiny macules. Looks normal.
1·12	÷	88·23	79·4	Do.	Almost complete disappearance of the shiny macules leaving very light hypopigmented macules. Considerable subsidence of infilt. of face.
4·0	1·0	70·59	82·35	Do.	Marked clinical imp. considerable subsidence of infilt. and nodules on ears cheeks and chin.
÷	÷	70·59	76·47	Do.	Almost complete subsidence of the infiltration. Macules looks normal.
1·75	÷	73·53	79·41	Do.	Almost complete subsidence of the infilt. macules. Looks normal.
3·31	0·75	79·41	85·29	Mild.	Marked appreciable clinical imp. considerable subsidence of the infilt. of face; nodules have markedly subsided. Encouraging result is noted.
3·81	0·75	73·53	85·29	Do.	Very marked clinical imp. considerable subsidence of all the lesions within this short period.

noticed a marked reduction of the B.I., yet the flat macules, either hypochromic or erythematous seemed to persist in a milder degree. In certain apparently similar cases the improvement was rapid but the why of it is not known. Of course, we *do* treat the predisposing causes.

Summary and Conclusions.—The results vary in different groups. (1) Those on a lower dosage show much less tendency to

pass into a state of reaction. The results in the low-dose-groups are not unsatisfactory. (2) The drugs affect both tuberculoid and lepromatous leprosy and also indeterminate cases. (3) Early treatment is definitely desirable and beneficial. No case treated early, became severe. The earlier the treatment, the greater the chances of arrest. (4) Children seem to tolerate bigger doses, especially of DDS. (5) Pregnant patients seem to tolerate well, a dose of 600 mg. per week, (Table II. DDS group, Case No. 11). Pregnancy does not appear to be a contraindication.

A word of warning.—There is danger in outpatients misusing DDS in oral administration. The patient should not be allowed to take the tablet (DDS) without consulting the doctor who is alone competent to decide if a patient may safely take it or not.

I wish to place on record my grateful appreciation of the help and advice given to me by Dr. R. G. Cochrane and Dr. K. Ramanujam, without whose co-operation I would not have been able to carry out this experimental study.

Dr. Cochrane's Report of his Visit to the Pan-American Leprosy Conference

During his tour Dr. R. G. Cochrane visited the Institute Oswaldo Cruz (Rio-de-Janeiro) and discussed with Dr. Souza-Araujo, the latter's experiments on the growth of Myco. lepræ and subsequent inoculation into animals. It was apparent that the South American workers were describing under the division of tuberculoid leprosy two conditions, namely reactional tuberculoid (with a negative or weak positive lepromin test) and tuberculoid in reaction (strongly positive lepromin test). At Sao Paulo, he saw the work of Dr. Nelson de Souza Campos and concluded that there seemed to be little doubt that BCG vaccination would change a lepromin negative to a lepromin positive (Mitsuda) and this positivity of the lepromin had been maintained in many instances for 2 years, although the tuberculin allergy after BCG vaccination changed over to the negative side after one year. At the Buenos Aires Conference two main topics were dealt with, which were of widespread interest to those studying leprosy *viz*: (1) classification and the possibility of the change of type and (2) sulphone therapy. There was general agreement that the indeterminate group of cases divided themselves into (*a*) those with a histology showing simple inflammatory changes (uncharacteristic histology, pre-lepromatous lesions, simple macular lesions of the Cairo classification, and resolved lesions) and (*b*) those with a histology showing granulomatous changes (borderline, reactional tuberculoid etc.). Dr. Payne's paper to the Conference was of interest with reference to two particular points *viz.*, (1) that the action of drugs on the two mycobacteria of tuberculosis and leprosy may not be parallel and that (2) the mono-substituted derivatives were probably more active than the di-substituted ones or even the parent substance DDS.—(*Leprosy in India*, 25: 1, Jan. 1953, p. 35).

LEPRA-REACTION AND ITS CONTROL

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and Specialist in Tuberculosis, "Cochin Villa," Kumbakonam.

LEPROSY is a disease having many peculiarities which distinguish it from other skin diseases. The main peculiarity of the disease lies in its low toxicity, long latent period, insidious onset and prolonged duration. The essential characteristic in which it differs from all other bacillary diseases is its particular affinity for the peripheral nerves. The delayed tissue reaction which limits the infection in some cases, divides the disease into two essentially main types *viz.*, (*a*) Tuberculoid and (*b*) Lepromatous. Leprosy, is a social and a physical disease and we have to bear this in mind in planning and adopting suitable measures of treatment and control.

I shall deal in this article, not with the signs and symptoms of leprosy nor with its classification, but only with the main points relating to the lepra-reaction and its control.

The main clinical signs are to be seen in the skin and mucous membranes and to some extent in the peripheral nerves. The signs of leprosy depend on the local reaction of the tissues to the invading bacilli. Skin lesions appear in proportion to the distribution of the bacilli. Next to the skin, the mucous membranes of the nose, mouth, pharynx, and respiratory passages are affected, especially in the lepromatous type. The mouth seldom shows signs, before the skin becomes severely affected; later on the palate and tongue are affected, only to be followed by nodulation. Involvement of the fauces and glottis causes contraction, giving rise to hoarse voice which is called "leper whisper" and to dyspnœa. So far the testis is the only internal organ known to be attacked, slowly getting atrophied, followed by gynæcomastia. Though cases of lung leprosy have been reported from the Phillipines, confirmation is lacking.

Lepra reaction is characterised by the acute exacerbation of the disease which comes on suddenly especially in lepromatous conditions, and is associated with general malaise, fever, local signs of inflammation œdema and general weakness, restlessness and insomnia. True lepra-reaction occurs only in lepromatous cases. The reaction does not occur before the infection becomes generalised and well-advanced. It is chiefly in the lesions of the skin that the local inflammation begins, later spreads to mucous membranes and at a later stage spreads to some extent to the peripheral nerves. Associated signs of swelling of the testis, enlargement of the liver and spleen, pain in the bones, synovitis, severe arthritis, affections of the eye, epistaxis, tingling neuritis, high pyrexia resembling malaria, typhoid and septicæmia may or may not be present; some cases develop rashes often mistaken for eruptive fever. In some cases we get diarrhœa, dysenteric stools, vomiting, epigastric pain, tympanites, otitis, mastoiditis and inflammation of the lacrimal sac.

In the female, severe hip-pain, abdominal pain, dysmenorrhœa and vomiting are also present. The entire body area may get simultaneously involved, or the reaction may localise itself in one single lesion which may later on spread to other parts. Different layers of the skin and subcutaneous tissues may react at the same time or the deeper layer may react first, and the superficial later on. The reaction may be slight and of short duration, or it may be severe, with high pyrexia lasting for weeks or months, or it may be recurrent—*i.e.* symptoms may appear and disappear, returning at regular or irregular intervals, or progress continually without yielding to any treatment, and ultimately reach the fatal termination. We get in very severe forms, exfoliation of the epidermis, abscess formation in the nodules and indurated lesions of the skin. There may occur in some cases, spontaneous cure by the bursting of the abscess and healing up, or extensive ulcers of varied types may appear which heal with difficulty ; the general poor health and the superimposed secondary infections often go to add to the bad prognosis of the condition. There may or may not be concurrent reaction of the mucous membranes with inflammation and ulceration in the nasal mucous membrane, swelling of the glottis, and nodules in the trachea which burst and discharge pus. We often see ulceration of the tongue, palate and faucies also. In some cases the eyes are affected, leading to conjunctivitis and keratitis.

The signs and symptoms depend on the severity of the reaction, its duration, the general health, and vitality of the patient and the other inter-current infections, such as infestation with worms, dysentery, tuberculosis and skin conditions, which often take a chronic turn. I have seen cases of reaction with and without fever in this hospital. It is noteworthy that the lepra-reaction *does* occur even in patients whose general health is good though it is more common in persons who show some deterioration in general health. The mechanism of the lepra-reaction seems to be obscure. Opinions differ on this point, and it is not possible in this short paper to discuss the various theories and findings of different workers. It is very likely however, that allergy plays an important part in the lepra-reaction, though its precise mode of action is not clearly known. The result of the administration of Antistine, Benadryl, Adrenaline and other anti-allergic drugs shows that, to a certain extent, allergy does play an important part in the mechanism of the lepra-reaction. But according to Dr. Cochrane, one of the leading world authorities on the subject, this theory cannot be sustained as there cannot be an allergic process without evident signs of allergy.

The role of sepsis—especially the presence of septic organisms, in the devitalised skin or in open wounds which complicates and aggravates the reaction—is always to be borne in mind. I have seen patients with plenty of PA. having severe reaction. The reaction sometimes occurs in an epidemic form, spreads like erisypelas from patient to patient. I have seen cases remaining always on the

verge of reaction ; any little indiscretion in diet, exertion, worry, or change in climate is enough to transform a mild reaction into a severe type. It has been suggested that lepra-reaction results from the intensive mobilisation of macrophages in an attempt to dispose of the bacilli which have suddenly multiplied. This results in an excessive stimulation of the reticuloendothelial system, an upsetting of the fine equilibrium between the parasite and its host and a setting up of a febrile condition which continues as long as this equilibrium remains disturbed.

The lepra-reaction appears to be a defensive process but being always unsuccessful, it does more harm than good to the patient. The lepra-reaction in its severe form is one of the most distressing complications of leprosy and calls for careful and skilled treatment.

Treatment i.e., control :—As a routine, all cases are first treated with plenty of alkalies orally and intravenous injections of Potassium or Sodium Antimony Tartarate 2 c.c. of a 2% solution every other day for six injections. The injections if continued indefinitely will produce an adverse effect, and make the condition worse. Many cases do not yield to this treatment, and hence there is the necessity for changing the drugs as and when required.

Antimony combined with calcium gluconate 10% in 10 c.c. doses is found to benefit in some cases. *Stibinol* '100' a product of the Brahmachari Institute at Calcutta, is an Antimony gluconate preparation, which gives satisfactory results and is easy of administration. Fantorin and Anthiomaline are some of the other Antimony preparations I have been using in the case of patients who can afford to buy them.

The intravenous injection of 20 c.c. of a 2% solution of calcium gluconate daily for ten days has given good results where antimony had failed.

10 c.c. of glucose 20% solution I.V. daily for six days has been of use in chronic cases showing signs of septicæmia.

10 c.c. Calcium gluconate 10% combined with 200 mg. Vitamin C does good in the few cases with general emaciation and lowered vitality. Calcium gluconate with Vitamin C (Sandoz) or Calcinol with Vitamin C (Raptakos) are some of the proprietary products of this type which give good results.

A few injections of Vitamin B_{12} intramuscularly have been found useful in chronic and persistent cases of reaction, particulary among those receiving sulphone therapy.

A course of six intravenous injections of mercurochrome 1% solution starting with 3 c.c., gradually increased to 8 c.c. given bi-weekly, has been found very useful in cases with exfoliative dermatitis. Traces of albumin may be present in the urine. It is not however, a contraindication for the use of the above drug.

Thiamine hydrochloride of Vitamin B complex (T.C.F.) 1 c.c. I.M. daily or on alternate days for ten injections and Elixir

B Vitamin (Albert David & Co.) have given good results in cases with severe inflammation of the peripheral nerves, especially those of the ulnar, just above the elbow joint.

An injection of 1 c.c. Adrenaline or 1% Novocaine 1 c.c. or absolute alcohol ½ c.c. into the inflamed nerve sheath, followed by the application of ice over the area, often dramatically relieves the intensive nerve pain.

In cases where the eyes are affected, frequent dilatation of the pupils with atropine and sub-conjunctival injections of normal saline or penicillin together with applications of penicillin or Irgafen ointment have given excellent results. Prompt treatment on these lines has saved many patients from getting completely blind. Small doses of sulphetrone will also help to clear eye-lesions as also other symptoms of chronic reaction.

When all the above drugs failed to control the reaction, I tried as an experimental measure, streptomycin 0·5 gm. daily or every other day for ten injections (observing the usual precautions) in a number of cases with gratifying results. It is not possible however, at this stage to draw conclusions of value as regards the benefit derived from its use in the comparatively small number of cases treated. Much more work will be necessary before one can confidently recommend it for routine use.

* Irgapyrin (Geigy) was used by me extensively in my cases with dramatic response. Its anti-pyretic effect was so good that the temperature touched normal even after the first injection in the majority of cases. Generally not more than three to five injections were necessary to control the reaction effectively. In my opinion, this is the drug of choice for treating lepra-reaction, if the patient can afford to pay for it. I have also received good reports about this drug from my assistant who used it in his private practice. Albuminuria is a contraindication for this useful drug. A report on the results of using Irgapyrin in this hospital is under preparation and will be published in the near future.

Some cases of reaction were controlled by auto-hæmotherapy but the results cannot be considered sufficiently encouraging to warrant extended trials. Good diet, careful nursing and environment add greatly to the effectiveness of the control of this condition, in addition to the therapeutic measures outlined above.

My thanks are due to Rev. Mother Paul, the Lady Superintendent of the institution for permitting me to carry out my clinical trials, and also to Dr. C. I. Pereira, the Bacteriologist and my personal assistant for his valuable help in furnishing laboratory reports and also for his valuable help in the preparation of this paper.

References:

1. Muir, E.—Manual of Leprosy. 2. Cochrane, R. G.—Pract. Text Book of Leprosy.

* A generous supply of this drug was made by M/s Amalgamated Chemicals and Dye Stuffs Co. for clinical trials.

INTRAVENOUS IRON IN LEPROSY

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THE effect of DDS (Diaminodiphenylsulphone) in leprosy has been extensively studied. Its effect on hæmoglobin and R.B.Cs. has also been studied and it has been shown that though there is almost a certain deterioration in both, at the beginning of the treatment[1], recovery follows and the initial level is reached in the course of a short time. Hæmotonics which were thought to be essential at the start of the treatment could easily be omitted if the doses were kept limited to the sub-toxic effective dose, which in most cases is 100 mg. a day. But it has also been seen that there are some cases which seldom improve with this effective treatment and the hæmoglobin level remains low and unaltered with various hæmotonics along with the stoppage of treatment with DDS. These cases with low hæmoglobin can seldom tolerate DDS treatment and thereby improvement in their conditions can never come in. Intravenous iron therapy was suggested in these cases and the result in 5 cases is given in this brief report. The experiment is in progress and more cases are being gradually added. Detailed blood examination could not be carried out for various reasons.

Cases:—Selection of cases was limited to only those who had taken plenty of iron orally and also liver without any appreciable improvement of hæmoglobin percentage.

Doses:—The initial doses were 2·5 c.c. to 5 c.c. intravenously on alternate days for 3 to 4 injections and then 2 or 3 injections of 10 c.c. each at an interval of 10 days were given (1 c.c. contained 20 mg. of saccharated iron).

CASE REPORTS.—1. N., female, aged 30 years, L_3, was put on DDS injection in Aug. '50 and the treatment was given twice weekly. In Aug. '51 she started reacting and the hæmoglobin went down to 44 (Sahli). Inspite of oral iron therapy in big doses from 17th April to 20th September, her hæmoglobin could hardly be raised over 52. All possible care was taken to remove the causes of anæmia including the stopping of anti-leprosy treatment but without effect. She could not stand the DDS treatment during this period.

I decided to put her on intravenous iron therapy (Iviron) and her condition improved and R.B.C. rose from 3120000 to 4250000 per c.mm. and the hæmoglobin from 52% to 64%. She has been standing the treatment well now.

2. G., male aged 23, advanced L_3 case was put on DDS tablets in Aug. '50. Inspite of all possible care against the causes of anæmia and giving iron tablets and other hæmotonics by mouth, his hæmoglobin percentage could not be improved more than 50 to 54 and it now and then deteriorated to 44; but with iron intravenously

his condition improved to 62. The number of reactions has lessened and he has been showing signs of improvement.

Table showing the results of treatment, as shown by gradual improvement of Hæmoglobin after Iviron intravenously

Name	Date	RBC	WBC	HB%	Iviron dose
(1) N.	5-11-'52	3120000	6200	52	—
	6-11-'52	—	—	—	2·5 c.c.
	7-11-'52	—	—	54	—
	9-11-'52	—	—	54	5 c.c.
	11-11-'52	—	—	48	10 c.c.
	12-11-'52	P A T 2 c.c.		—	—
	20-11-'52	4190000	6350	52	—
	21-11-'52	—	—	—	10 c.c.
	28-11-'52	4360000	6850	52	—
	1-12-'52	—	—	—	10 c.c.
	11-12-'52	4250000	6500	52	10 c.c.
	16-12-'52	—	—	64	—
(2) G.	7-11-'52	3870000	7150	58	—
	8-11-'52	—	—	—	2·5 c.c.
	10-11-'52	—	—	58	5 c.c.
	12-11-'52	—	—	58	5 c.c.
	19-11-'52	3820000	6600	58	—
	22-11-'52	—	—	—	10 c.c.
	29-11-'52	4440000	—	58	—
	2-12-'52	4500000	5200	58	10 c.c.
	12-12-'52	—	—	—	10 c.c.
	16-12-'52	—	—	64	—
(3) L.	7-11-'52	2780000	8750	36	—
	8-11-'52	—	—	—	2·5 c.c.
	10-11-'52	—	—	42	5 c.c.
	12-11-'52	—	—	42	5 c.c.
	19-11-'52	3450000	9300	44	—
	22-11-'52	—	—	—	10 c.c.
	29-11-'52	3510000	8750	50	—
	2-12-'52	3850000	8050	54	—
	12-12-'52	—	—	—	10 c.c.
	16-12-'52	—	—	54	—
(4) A.	24-3-'53	2470000	—	44	—
	25-3-'53	—	—	—	2·5 c.c.
	27-3-'53	—	—	48	5 c.c.
	29-3-'53	—	—	40	5 c.c.
	5-4-'53	2410000	9600	44	—
	15-4-'53	—	—	—	10 c.c.
	18-4-'53	3970000	9000	48	—
(5) B.	24-3-'53	3540000	—	42	—
	25-3-'53	—	—	—	2·5 c.c.
	27-3-'53	—	—	50	5 c.c.
	29-3-'53	—	—	46	5 c.c.
	5-4-'53	3910000	4500	52	—
	15-4-'53	—	—	—	10 c.c.
	25-4-'53	4200000	5000	54	10 c.c.

Iviron—1 c.c. contains 20 mg. of saccharated iron.

3. L., male, aged 38, L_3, case in an advanced stage. He was put on DDS tablets in March '50. His Hb. was 60%; under continued DDS treatment his condition worsened and the Hb. went down

to 40% in Sept. '50. With vitamin B_{12} it went up to 54% for a short while but again deteriorated to 28 in May '51 when iron with B_{12} was prescribed was ultimately DDS treatment had to be stopped in Aug. '51 as the Hb. % went down to 28% again. This condition improved with addition of Folic acid to vitamin B_{12} and iron and DDS was started again. But the improvement did not last long and iron intravenously was advised in November '52, which improved the condition. He has been tolerating the treatment well ever since.

4. A., female, aged 50, a moderately infectious case. While on treatment with DDS her hæmoglobin went down and she reacted badly and could not stand the treatment; on giving Iviron her Hb.% improved only a little but the total R.B.C. count improved appreciably. She is much better now.

5. B., female, aged 30, a nodular case of leprosy had been treated with hydnocarpus oil from 1943–1950. When put on DDS treatment, her hæmoglobin became low. She was given iron and other hæmotonics with only temporary benefit; but on subsequent intravenous iron therapy, she has definitely improved and is standing the treatment well.

Discussion.—From the results recorded above it is observed that intravenous iron had a definite action on hæmoglobin % which deteriorated with DDS treatment. All the cases improved as regards hæmoglobin content. Case No. 4 did not improve much though her R.B.C. per c.mm. rose higher than in any other case. The total R.B.C. improved greatly in all cases.

Conclusion.—Intravenous iron therapy is valuable in cases, where oral administration of iron and any other hæmotonics fail to improve the condition, especially in cases of leprosy under DDS treatment.

Acknowledgement.—Thanks are due to:—Dr. Nair, M.D., F.R.C.S., for his kind suggestions, Messrs. Indian Schering Limited for a free supply of Iviron, and Mr. Andrew Bansriar for helping with the laboratory tests.

References:

1. Dharmendra—Leprosy in India, 1951, June, page 57.
2. Roy—Indian Medical Gazette, 1951, Aug., page 373.

Psychiatry Simplified

Dr. G. Gordon, a du Pont psychiatrist has a simple explanation for much of the mental sickness that plagues society :—"A well person put at bed-rest for weeks deteriorates physically. On the other hand, strength and stamina are greatly increased by work. The same applies to mental health. The most disabling sickness is found amongst those who have succeeded best in shirking responsibility".—(*Efficiency News*, July 1953).

BOOK REVIEWS

Human Parasites and Parasitic Diseases—By K. D. CHATTERJEE, M. D. (Cal.), Calcutta, India, 1952.

Hitherto we have had to rely on books written by western authors for information relating to tropical diseases and parasitic infections peculiar to the tropics. This was indeed a paradox. Dr. Chatterjee has remedied this serious defect by publishing this book which may be considered a standard treatise on the subject, which would help the students and practitioners and stimulate research on different aspects of the problem. The author has devoted 17 long years to produce this *magnum opus* which contains 750 pages; more than half of the book has been devoted to amœbiasis, kala-azar and malaria, and in the final chapter antihelminthics have been dealt with in detail.

The presentation of the various subjects covered by this book has been done in a masterly and interesting fashion, with the relevant references recorded in proper position.

The get up of the book is excellent as regards printing, the quality of the paper and the neatness of the diagrams which are self explanatory.

The book will certainly be of great use to medical students, post graduates, clinicians and public health workers.

Synopsis of Pathology—By ANDERSON, III Edition, 1952. St. Louis. The C. V. Mosby Company.

This is a well written book, in the seven hundred pages of which is epitomised a very large quantity of valuable information in such a way that the really most useful points contained in the author's larger Text Book of Pathology have been brought out prominently and with considerable effect. The references given at the end of each chapter are complete and should enable the research worker and post-graduate student to find the originals readily for detailed study.

NEWS AND NOTES

Terramycin Effective against Stubborn Skin Infections

One of the most extensive studies on the use of this antibiotic in the treatment of boils, acne, impetigo and other skin diseases was presented by members of the Department of Dermatology, University of Maryland at a recent meeting of the American Medical Association. The antibiotic, administered orally or used in ointment form in more than 1750 patients suffering from a wide variety of the skin infections, was found by Robinson to be "definitely beneficial" in 18 different skin conditions.

Similar effects with terramycin have been reported in various medical journals by other clinical groups. Wright and Tschan obtained highly encouraging results in a study of 755 patients with various skin diseases. Eighty-two of the 90 impetigo patients in this group were cured within one to 7 days by using this antibiotic. Andrews *et al*, after a two year study involving 384 acne cases stated that the antibiotic "has been especially beneficial and well tolerated". In a later study, by Barnard and Orens on 34 acne cases which failed to respond to other methods of treatment, not a single one failed to exhibit some degree of objective improvement after treatment with terramycin.

Two British skin specialists report the antibiotic also effective against boils and the infection commonly called barber's itch. Valentine and Hall Smith point out that in these diseases, each sore is surrounded by infection-causing organisms which are often carried by the hands to other parts of the body. To prevent reinfection, terramycin ointment was applied in the area of infection and in both nostrils. Recovery following this treatment was described as "impressive" and "rapid"–(*M.P.I.B. Bulletin*, June 1953).

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,, ,, 10mg 1 cc 6 amps box 4-4
,, Isonicotinic Aid Hydrazide :-
50mg 100 2-12; 1000 tabs bot 25-0
Dumex Isonex 100 tabs bot 3-5
Dihydrostreptomycin 1 gm.
Glaxo Merck Pfizer A.H Rhodia
1-13 1-10-0 1-14 1-10 1-10
Strepto with Penicillin :—
Belgium Rhodia Glaxo Squibb's
2-4 2-6 2-4 2-11
Pfizer Combiotic (Penstrep) 3-4
Procain Penicillin 4 lac:—
Dumex Glaxo Squibb Pfizer
1-0-3 1-1-6 1-7 1-14
Procain Penicillin 20 lac Glaxo 3-13-6
,, ,, 20 lac Dumex 3-10-6
Strepto 5 PAS. Lepetit 3-8
Penicillin Sodium Crys. G.
2 lac 5 lac 10 lac
Dumex 0-9-6 1-0-3 1-10-6
Glaxo 0-10-6 1-1-6 1-12-6
Penicillin Lozenges 20;1-1 50 2-0
,, Eye Ointment 1-2
,, Skin Ointment 1-6
Aureomycetin 8 cap. 19-0
Terramycetin 8 17-0 16 caps 32-8
P.D. Chloromycetin 12cap 17-4
,, ,, liquid 60 cc 14-4
,, Camoquin 3 tab. 0-10-0
,, Abedec drop 15cc 5-12
,, Ferradol USA 1 lb bot. 5-4
Quinine Sulph Jap. 35-8 How 52-8 lb.
,, ,, Jap. 1oz 3-0 ,, 4-0 oz.
,, Sulphate plain 2gr 100 tabs 2-4
,, ,, ,, 5gr 100 ,, 4-4
,, Bisulph How 5gr 1400 ,, 60-0
,, Bihydrochlor 2gr 100 ,, 2-12
,, ,, ,, 5gr 100 ,, 5-8
,, ,, 5gr 12 tabs How. 1-0
,, Bihydro 10grs 100 x 2cc
,, Ind. B.D.H. Evans B.W. P.D.
16-8 23-0 22-8 33-8 40-8
10-12 15-0 15-0 5gr 100x1cc
Cibazol 20's 1-10 250's 13-12
Bayer Atebrin 15 0-10; 300 7-4
,, ,, 1000 tab 10-0
Adopter Japan doz 2-4
Artery Forceps each 2-4
,, ,, ō Needle Holder ,, 3-4
Axiatraction Forceps 48-0
BDH Bismuth Carb 500 tabs tin 4-4
Berin 1 mg 25 tabs 0-8; 100 1-8
Breast Pump 1-8; Bistury 2-0
BW Atropine Sulph 1/100gr 20 tabs 0-10
BW Digitalin 1/100gr 20 tabs 0-8
,, Strichnia Hydro 1/60gr 20 tab 0-8
,, Hyocine Hydrobrom 1/100
or 1/200 gr 20 tabs 0-10
,, Nicotinamide 50mg 100x1cc 12-0
Calomel 1gr 5000 tabs bot 28-0
Catheter I.R. 0-8 Ger. 1-4
,, Metal for Male 1-8
Catheter Metal Female 1-8
Cork Screw ō Tin Cutter Eng 1-0
Ear and Nose Forceps each 3-8
Elasto Plast 2½ x 5 yds tin 2-0
,, ,, 3 x 5 yds ,, 3-0
Emetin Hydro ⅛gr 6x1cc PD 6-0
,, ,, 1 gr. 6 x 1cc ,, 9-0
,, ,, Endo ⅛gr 6x1cc 2-12
Enterovioform 20's 2-12; 100's 11-10
Ephedrin Hydro Ger ¼gr 1000tab 5-12
Eye Bath Glass each 0-10
F.L. Washable 0-5 each
,, ,, Crocodyle 0-8 ,,
,, Silvertex 2-4 doz
F.L. Durex Pkt. 1-12 Tin 2-0 doz
Fountain Pen Battery Comp. 3-0
Glass Pan 0-6; Glass Rods 0-3
,, Syringe 1 oz 0-8; 2 oz 0-10
,, ,, 4 oz 0-12
Gl. Macarbin 50 Micro 6x1cc. 5-8
,, Berin 50mg 10cc 3-2 100 mg 4-5
Glass Funnel 2" 1-4 3" 1-12; 4" 2-8
Head Mirrors c Cloth Band 13-8
Horse Hair 100 Strand box 2-0
Irgopyrin 5x5 9-8 50x5cc box 77-8
Hypo Syringe 50cc S.N.
Jap. 3-14; Italy 9-0; Germ. 8-8
Record Needle (Perfectum 5-8)
Jap Germ. Star. D.B.
1-12 1-12 4-0 4-8 doz
All Glass Needles Luer Mount
Jap 2-4;Ger 3-4;DB 5-0; BD 10-8
Hypo Syringe (SN Re. 1 more)
A.G. Jap 2 5 10 20 30cc
Superior 0-7 0-11 1-0 1-12 2-12
Italy 1-0 2-0 2-10 3-8 5-8
German 0-14 1-1 1-8 2-10 4-12
Record Ger 3-4 4-12 6-8 9-0 13-8
Boston 4-12 5-8 7-0 11-12 15-4
B.D. Lock 8-4 14-0 15-0 17-0 23-10
Japan ,, 1-8 2-8 3-4 4-4 7-0
Insulin Syringe each 5-0
Injection Eng :—
Camphor Ether c/oil 12x1cc 0-12
,, In oil 12x1cc 1-0 box
Mercury Biniodide 12x1cc 0-10
,, Iodine Rubrum 12x1cc 0-10
Sodi Glycero Phos. 12x1cc 0-12
Strychinine Hydro 12x1cc 0-12
Japan Parker Type Fountain Pen 3-8
Laxative Vegetable 100 tabs bot. 1-0
Leukoplaster 2½x5yds tin 1-8
,, 3x5 yds ,, 2-0
M&B 693 ·1 gm 6 amps box 3-0
M&B Praequine 500 tabs bot 1-8
,, Santonine 1 dr. dr. 4-4
NAB 15's 0-10; 30 0-11; 45 0-13
Nicotinic Acid 500 1-13 [6.0-15
Microscopic Glass Slide doz. 0-8
Neosalvarsan 0-15 ·30 ·45 ·60grm
(·90gm 1-4) 0-14 0-15 1-1 1-3
Oil Chinopodium 1 oz oz. 4-8
Oint Slab Graduates each 4-4
Ostocalcium 50 tabs. bot. 2-1
Stearn's Bicolate tab. bot. 3-12
Ointment:-Acid Boric USA 1oz 0-4 each
Oibazol Oint ,, 2-6 ,,
Acriflavin Boot's 2 oz 0-6 ,,
Atropine ,, ½ oz 0-12 ,,
Burnol Boot's ,, 1-3 ,,
Blue USA ½ oz 0-5 ,,
Golden Eye Oint. 0-12 ,,
Calomel USA 1 oz 0-8 ,,
Gentian Violet Jelly 4 oz 0-8 ,,
Mercurial ½ oz 0-5 ,,
Sulphanilamide 4 oz 0-9 ,,
Santonine Calomel ½gr 100tabs 6-0
Pulsometer ½ min each 3-4
PAS German 100 gm ,, 5-8
,, ō Calcium Ger. 100gm ,, 5-12
,, ,, ,, Ger 7½gr 100 tabs 3-8
,, ,, ,, ,, 7½ gr 250 tab 7-8
Potas Chloras 500 tab. bot. 3-8
Pessary Ring 0-6; Check 0-8
,, Hodges Vul. 0-10
Plastic Gly. Syringe 1oz 3-0 2oz 4-4
Resochin tab 10 1-12; 100 14-8
Rubber Gloves '7½ or 8' 0-12 pair
Roche's Beflavit 250 tab bot 1-4
,, Beflavit 1mg 50x2cc box 1-4
Sulphagaisidine Boots 500 tabs 11-8
,, thiazole ,, 500 ,, 19-4
Sulphanilamide 1000 tab 11-0
,, gunadine 500 10-12 1000 20-8
,, diazine 500 37-8 1000 74-0
Sulphamezathine 3cc 25amp 6-8
,, 100 tab 6-14; 500 tab 28-8
Suture Needle Eng. 0-4 each
Saline App. 300cc 8-0; 500cc 10-8
,, Needle 1-12; NAIL CUTTER USA 1-8
Serum Needle 2" 0-12; 3 " 1-0
Silk Worm Gut 100 Strands box 1-8
,, Ligature Hank 1-4
Spirit Lamp Glass 2oz 1-10; 4oz 1-14
,, ,, Metal 2oz 2-4; 4oz 3-8
Spatula Bolus 4" 5" 6" 8"
0-14 1-0 1-2 1-4
Sterilizer Nickel 6" 13-8; 8" 20-8
Stethescope Ger B.D. Type 12-8
Test Tube Stand for 12 tube 3-0
,, ,, Holder NP c Handle 2-4
,, ,, Assorted each 0-3
Tongue Depressor 1-4 Folding 2-0
Tourniquet in tin 2-8
Vaginal Speculum Folding 9-12
Thermometer Germ. 0-15; Jap 0-10
,, Zeal 2-4; USA 1-4; Eng 1-4
,, Hicks 3-8; Jap Flat 1-4
TCF. Vit. B Complex 10cc 5-0
,, W. Liver Ext. 10cc 3-2
,, ,, ,, c C & B 10cc 4-6
,, ,, ,, c Vit. B_{12} 10cc 4-12
USA Bandages Brown 3x6yds 0-6
,, ,, ,, 2x1½ yds 0-3
,, ,, white 3x6 yds 0-8
,, ,, First Aid 4 x 4½ ,, 0-4
,, Triangular 24x48 Ft. 1-0
,, Multivitamin 1000 tab bot 9-12
Vitamin B_1 USA 500 tab. 1-0
,, C Eng. 50mg. 1000 tab 16-8
Weight set Dr. & Gr. set 0-12

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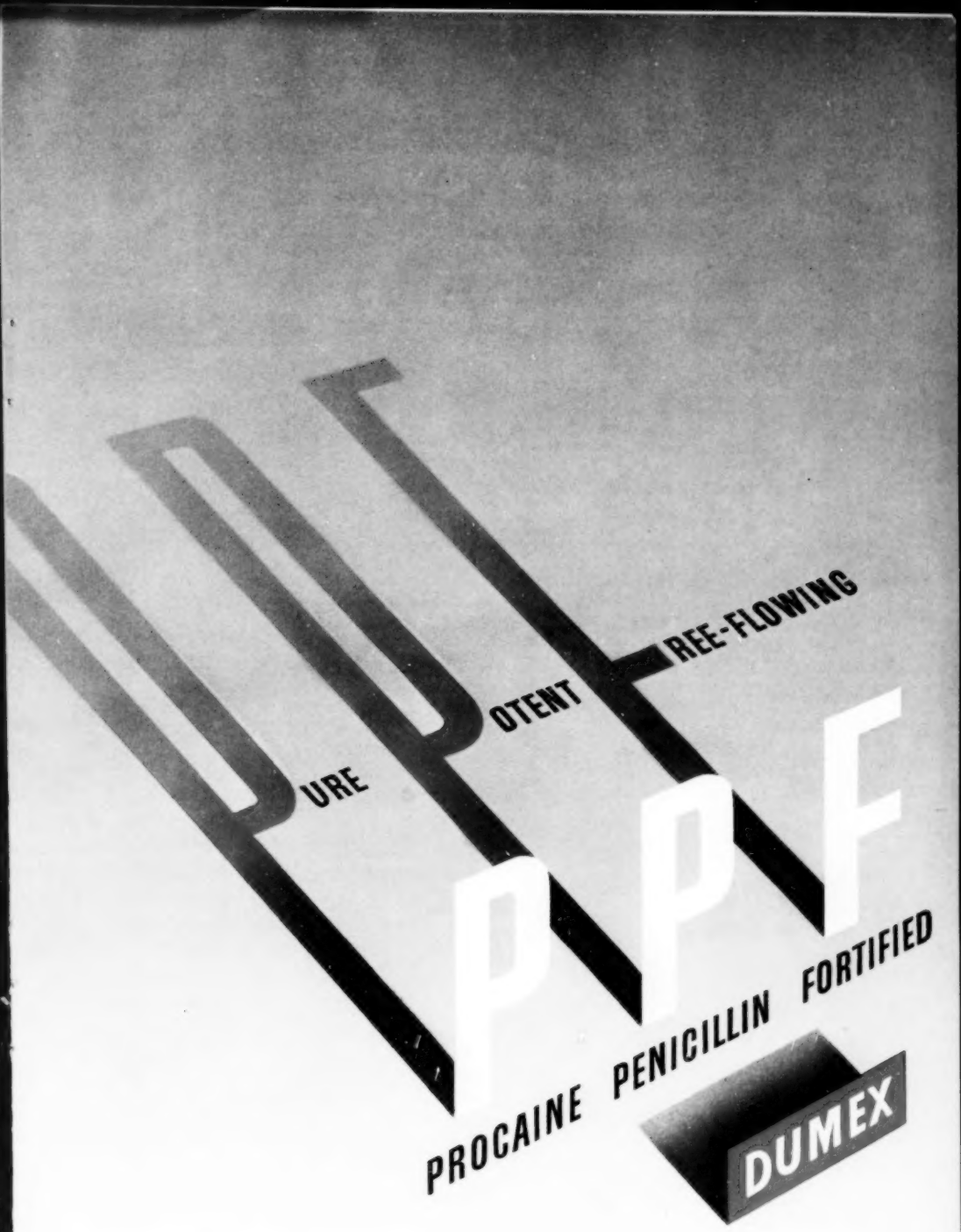
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the liver extract
with minerals
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Ampoules of 5 and 10 cc.

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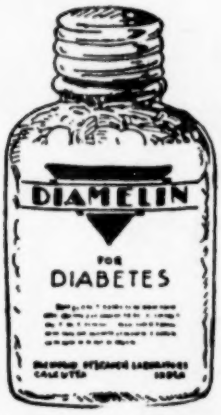

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Phenobarbital	... 1/6 gr.
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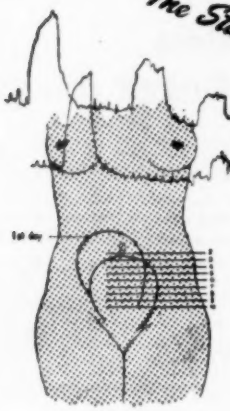

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[1] Britton C. J. C. (1950): Practitioner, 164, 458

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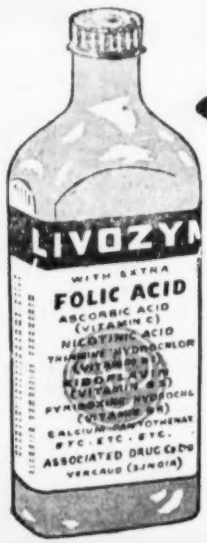

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OF VARIOUS KINDS FROM FUNCTIONAL & GLANDULAR DISORDERS

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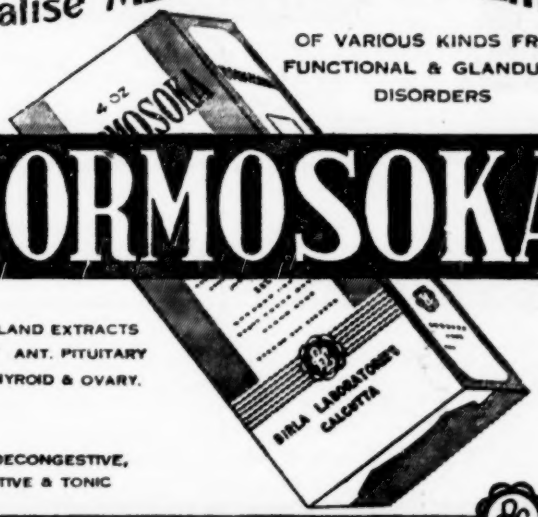

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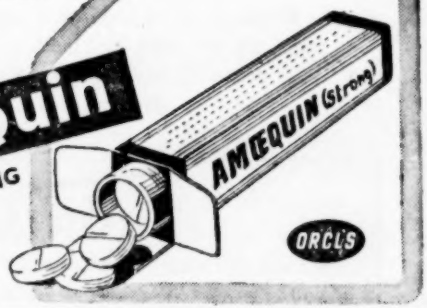

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Acid Boric 0-12 Pot. Citras
Menthol 2-12 [2-12 lb
Codein Phos 5-0 Dionine 6-12 dr
Ext Ergot Liqd. 4oz 4-4 Eng 9-8
Oil Chinapodium oz 4-12
Aletris rio 11-0 Eng. 2-6
Aspirin 3-12 Sod Salicylas 3-12

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■ needs no refrigeration.

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300,000 units—crystalline procaine penicillin G
100,000 units—buffered crystalline sodium penicillin G
1 Gm.—dihydrostreptomycin (as the sulfate) for 3 cc. dose.

Combiotic 0·5 Gm. Formula consists of ..
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100,000 units buffered crystalline sodium penicillin G
0,5 Gm.—dihydrostreptomycin (as the sulfate) for two-cc. dose.

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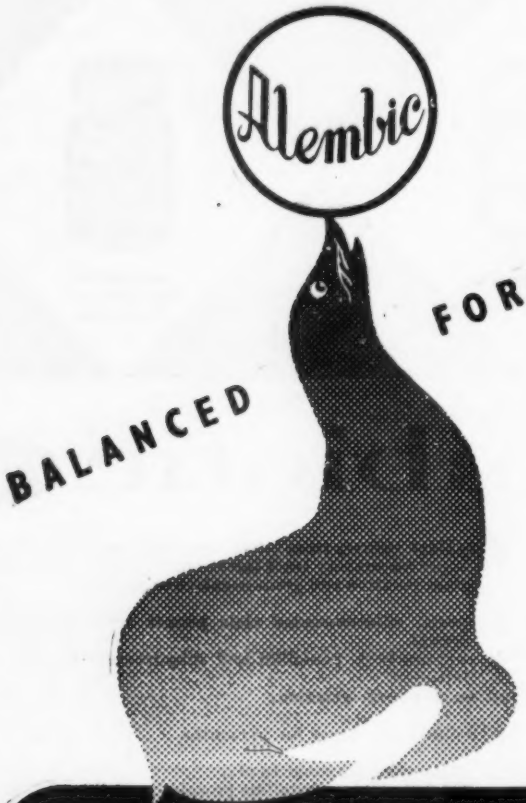

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Auromycitin 8 Cap. 19-0 [14-4
Terramycin 8 Cap. 16-12; 16 29-8
Combiotic P.S Pfizer 3-2
Pan-Strepto Merck 2-14
P. D. Camequin Tab. 0-10; Combex
Penicillin G Crys. [10cc. 6-12
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,, — 0-9-6 1-0-3 1-10-6 Dumex
Dihydro Strepto 1gr Pfizer 2-1
Merck, Belg 1-10; Rhodia 1-9
Procain Penicillin 20 lacsGl. 3-14
,, ,, ,,Dumex 3-10-6
Dumex Pf. Gl.
4 lacs 1-0-0 1-13-0 1-1-0
3 lacs x 10 cc. Oily Merck 8-6
Penicillin Skin Oint 1-6 Eye 0-15
,, Lozenges 500 10-0 20 1-0
PAS Tab. Eng. 100 3-8; 250 7-12
,, Ca. 100 3-2; 250 7-8; 500 15-8
P.A.S. Dumex 100 grm 7-4 Itl. 3-4
Quinine Jap. 34-4; Java 44-8
,, Roche 49-0; Howds 49-8
,, oz Jap. 2-14; Howds 3-10
Q. Bihydro Amps. 100x10grsx2cc.
,, Ind. B D.H. Evans B.W. P.D.
,, 15-0 22-0 22-0 32-8 38-0
,, 10-12 14-8 14-8 100 15gr.x1cc.
Euquinine Howd. 5-8; Java 4-7
,, Roche 6-14; Jap. 3-13
Q. Tab. 2grs 100 2-12; 5gr 5-0 How
,, ,, 5 gr. 1400 How. 54-8
,,Bihydro2grs 100 2-10; 5gr 5-4 How
Pamaquinine 300 Tab. 0-12
Asparine 1000 Eng. 5-14; Ind. 4-8
Mepacrine Eng 1000 10-0; ICI 11-12
Quinacrine MB 500 6-0
,, USA. 5000 48-0; tin 1000 10-4
Ephedrin ½ Gr. 1000 5-10 Germ.
Yeast Tab. Eng. 5gr 4-6; 7½gr 5-4
Soda Mint ,, 2-10; Ind. 2-2
Paludrin 1000 x 1 gr. x 23-8
,, 3 grm x 500 25-0; 1000 49-8
Ext. Ergot. 4oz 4-0 Saridon 250 25-0
Potas Chloras 1lb. 3-0;tab. 1000 6-0
H.T.Ind. Morph. Sul ½ gr x 20 2-8
,, P.D. ,, ½ gr x 20 3-0
,, BW ,, c Atropine ½ gr x 20 4-0
Ind. Morph Sulph ½gr 12 amps 5-8
Sulphamezethin 25 amp. 6-4
Multivitamin tab. USA 1000 8-12
Santonin Synthetic German
1 dr. 2-14· 1 oz. 19-0 [1000 21-0
Vit. B Complex tab Orig. USA
TCF. Vit. B Complex 6x 2cc 6-0
,, Vit. 10cc 4-14; 1x25x1cc 12-4
TCF. W. Liver Ext. 10 cc 3-0
,, ,, ,, c C & B 10cc 4-4
,, ,, ,, c Vit. B_{12} 4-10
Vitamin K 100 amp BW 17-8
Milk with Iodine amp. 100x5cc. 13-0
Aletaris Eng 2-4; Rio 11-5
Riscchin tab. 10 1-12; 100 13-14
Redoxon 6x2cc. 4-12; 50x2cc. 36-0
,, 3x5cc. 4-4; 25 x 5cc. 27-12
Merck H.T. Emetin ½ gr. or1 gr.3-10
Sulpha Tab. 1000 500
,, nilamide Eng. 10-8 5-6
,, guinidine Hol. 18-0 10-4
,, Boots 500 11-4 MB 13-4
,, thiazole Boots 19-4
,, mezathine (100 6-14) 28-0
,, diazine Eng. 71-0; MB 40-0
Sulphatrone (100 10-0) 41-4
Sulphatriad MB (100 8-8) 41-8
Sulphonamide Bayer 1 lb. 7-4
,, cream 4 oz. Lilly 6-0 doz
Gentian Violet Jelly 4 oz. Lilly 6-0
Emetine amps. BDH ½gr.x12 7-8,,
,, 1 gr.x12 12-12; ½ gr. x 25 13-0
,, Endo 6 x ½ gr. 2-10
,, Emetine amp Ger. 12x1gr 10-8
,, P.D. ½gr.x 6A 6-0 1 gr. 9-0
,, B.W. ½ gr. 8-8; 1 gr. 7-12
Vit. B_{12} 100 micro Belgium
10cc 5-14; 5 cc 3-10
50 micro ,, 3-10; ,, 2-8
Vit. B_1 100 mg 10cc 3-4;
,, C 500 mg x 5cc x 5 A 3-6
,, ,, ,, ,, 30 A 18-0
Liver Ext. 10 cc Belgium
2 USP 2-10; 5 USP 3-14; 10 USP 6-0
Cibazol 250's 13-8; 20's 1-10
MB 760 27-0; MB 693 500's 34-0
Vitamin B tab. U.S.A.500 1-0
Liver Ext. 10cc. 2 USP P.D. 3-12
,, ,, 5 USP P.D. 7-12
Campolon 5x2cc 5-8; 25x2cc 25-0
Cal. Glu. 10% x 10 cc. 100 13-0
Glucose Sol. 25% x 25cc. x 50 8-8
Atophanyl I.V. Ger. 5-0; I.M. 5-0
Serin 50mg. 3-2; 100 mg. 4-6
Calci Ostelin 15cc. 3-3 Entedas 8-0
NAB. 15's 0-10; 30 0-11; 45 0-13;
Nicotinic Acid 500 2-0 [6 0-15
Acetylarsan Adult 6-6 Child 4-12
Atebrine Bayer 15 0-13; 300 7-12
Distil Water 100 x 5 cc. 4-8
,, ,, 10cc. 6-4; 2cc. 3-12
Normal Saline 100x5 cc 5-12
Sil. Vit. France 3-0; Protargol
Ethyl Chl. 100grm. Ger. 2-4 [1-12
Sedasaly 3-12 lb. Santonine dr. 4-2
Calomal oz. 1-0; Chlorobutol 1-6
Menthol 2-12; Gentian Violet 1-0
Thermometer Germ. 0-15; Jap. 0-10
,, Zeal 2-4; USA 1-3; Eng .1-4
,, Hicks 3-8; Becon Flat 1-8
B.D. Stethescope 21-8 Tripal 35-0
Becon Stethescope (B.D. type) 7-12
Plastic tubing 1-4; Rubber 0-12 yd.
Erkameter 72-0; Aspirin 3-12 lb.
Detecto Weighing Machine 49-8
Saline Apparat. comp 300cc. 7-8
Vincarnis large 8-8 [500 cc. 10-8
Wall Thermometer Japan 1-12
Sandoz. Cal. Gluco ~ Vit C.
5 x 10 c.c. 6-8;10x5 c.c. 10-14
Sandoz Cal.Glu.10% x10cc 5amps 5-2
,, ,, ,, ,, 5 c.c. x 10 8-12
,, 50 amp. 44-12 10c.c.x 20 19-6
Entrevioform 20's 2-14; 100's 11-8
Abs. Cotton 1-10; Lint 3-4
Abs. Gauze 18 yds x 25" lb. 4-4
Bandages 3½ yds x1" to 6" 0-8
Hot water bag 3-4; Ice bag 1-2
Hypo. Syringe (S.N. Re. 1 more)
A.G. Jap. 2 5 10 20 30cc.
0-7 0-11 0-14 1-4 2-0
Italy 1-0 1-10 2-4 3-4 5-8
Germ. 0-14 1-1 1-8 2-8 5-4
Record Ger. 3-4 4-12 6-8 9-0 12-0
,, Italy 3-2 4-4 6-0 8-8 10-8
,,Comp. 6-0 9-0 11-0 — Germ.
Boston 4-6 5-0 6-8 9-12 13-4
B.D. Lock 8-4 14-0 15-0 17-0 23-10
Japan ,, 1-8 2-8 3-0 4-0 5-4
,, H.case 2-4 3-4 4-4 6-8 —
Hypo Syringe 50 cc. S.N.
Jap. 3-12; Italy 8-0; Germ. 8-12
B.D. Luer Lock 32-0; Jap. 8-0
Record Ger. 20-8; Italy 18-0
Record Needle (Perfectum 5-0)
Jap. Germ. Star. D.B.
1-10 1-10 3-12 4-0 Dz.
All Glass Needles Luer Mount
Jap. 2-10 Ger. 3-4 D.B. 4-12 B.D. 10-0
Atebrin Amp 3 grm 25 amp 15-4
,, 0.1 grm. 6 3-6 50 amp 25-0
Camphor-in-Oil 3 gr. x 1c.c.x100
,, ,, 3grs 12A Evans 1-0 [Cipla 3-12
Omnopon Amps with Needle
Irgapyrin 5 amp 9-4 [Tube 0-8
F.L. Durex Tin. 2-0 doz. Pkt. 1-12
Ear, Metal Syringe 2oz. 5-0;
Waterbury Co. 5-10 bot. [4 oz. 6-0
Oil Chinapodium oz. Eng. 4-12
Disp. Scale Nick 5-4; Brass 4-0
Gynomin tab 2-0 tube Spiton 3-0
Quinacrin amps 0.3grm tube 9-12
Nivaquin 10's 1-10
Codina Phos 5-0 Dionine 7-0dr.
Cafina Citrus 1-8 Bis Carb oz 2-0
F.L. Washable Germ. each 1-8
Vit. c tab 50mg Eng. 1000 15-0
Paludrin 5A 3-0 25A 11-4
Stethescope Pouche Plastic 1-8
Fountain Pen Battery China 3-0
Artery Forceps 2-2 Scalpel 1-12
Dress ,, 1-4; Tongue Depresser 1-4
Sterilizer 6" 12-8; 8" 18-8
Universal Tooth Forcep Sup. 4-8

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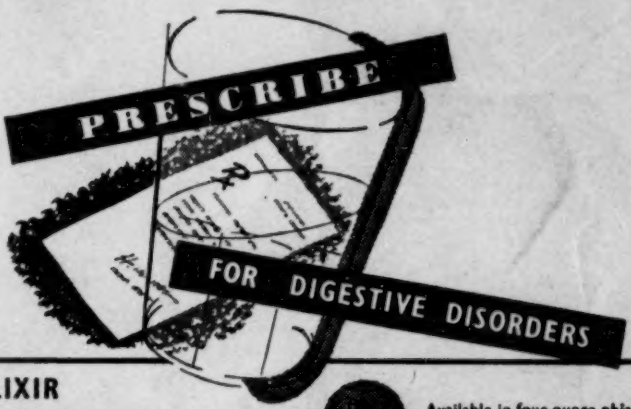

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